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## Table of Contents

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**ORIGINAL ARTICLES—** PAGE.

"Some Crippling Conditions and Their Treatment," by L. O. BETTS, M.B., B.S. .. .. .. .. .. 488
"Cardiac Dyspnœa," by F. R. FRASER, M.D., F.R.C.P. 491
"Ulnar Paralysis: A New Operation," by LOCKYER POTTER, M.D. .. .. .. .. .. .. .. .. .. 498
"Enuresis," by EDGAR H. M. STEHPEN, M.B., Ch.M. 502
"Appendicitis in Children," by P. L. HIPSLEY, M.D., F.C.S.A. .. .. .. .. .. .. .. .. .. 503

**REVIEWS—**

Errors in Refraction and Their Correction .. 505
Dietetics .. .. .. .. .. .. .. .. .. .. .. 506
A Bacteriological Manual .. .. .. .. .. .. 506
Present Day Knowledge on Tobacco .. .. .. 506

**LEADING ARTICLES—**

The Annual Meeting of Delegates .. .. .. .. 507

**CURRENT COMMENT—**

Exercise and Digestion .. .. .. .. .. .. 508

**ABSTRACTS FROM CURRENT MEDICAL LITERATURE—**

Surgery .. .. .. .. .. .. .. .. .. .. .. 510

**BRITISH MEDICAL ASSOCIATION NEWS—** PAGE.

Scientific .. .. .. .. .. .. .. .. .. .. 512
Medico-Political .. .. .. .. .. .. .. .. 513

**CORRESPONDENCE—**

Fees for Life Insurance Examination .. .. .. 513

**UNIVERSITY INTELLIGENCE—**

The University of Melbourne .. .. .. .. .. 513

**CORRIGENDUM** .. .. .. .. .. .. .. .. .. 513

**A HOLIDAY EXPEDITION** .. .. .. .. .. .. 513

**BOOKS RECEIVED** .. .. .. .. .. .. .. .. 514

**DIARY FOR THE MONTH** .. .. .. .. .. .. 514

**MEDICAL APPOINTMENTS** .. .. .. .. .. .. 514

**MEDICAL APPOINTMENTS VACANT, ETC.** .. .. 514

**MEDICAL APPOINTMENTS: IMPORTANT NOTICE** 514

**EDITORIAL NOTICES** .. .. .. .. .. .. .. 514

## SOME CRIPPLING CONDITIONS AND THEIR TREATMENT.[1]

By L. O. Betts, M.B., B.S. (Adelaide),
*Honorary Assistant Surgeon, Children's Hospital, Adelaide.*

It is my intention to discuss several conditions the early diagnosis and treatment of which are of importance. This paper is based mainly on work done in orthopædic clinics in England where these conditions are commonly seen.

### Wrist Injuries.

Injury to the wrist from a fall on the oustretched hand is one of the most common met with in practice. Bad results from these injuries are common in orthopædic departments. As these are avoidable, I would draw your attention to some of them.

Malunited Colles's fracture is due in almost all cases to failure in the one essential of treatment, that is complete reduction of the fracture. To insure this a general anæsthetic should always be given and particular attention paid to the restoration of the concavity of the anterior surface of the radius and its projecting lip. Reduction is as a rule easily maintained and as long as finger movements are given early, the final result will be good, whether the fracture is splinted for ten days or three weeks.

An unreduced Colles's fracture should be broken down with the hands or with a small Thomas's wrench and reset. This can be done up to six weeks after the fracture; I have seen one as late as ten weeks. Tenderness of the callus is a sign that union is still unsound. Later an open osteotomy of the radius is the only means of correcting the deformity, if such be deemed wise. In coming to a decision in late cases we must remember that unreduced Colles's fractures, although often giving moderately good functional results at first, will later frequently be followed by a weak, painful wrist with a chronic teno-synovitis, especially if taxed with heavy work.

Two carpal injuries, fracture of the scaphoid and dislocation of the semilunar demand our attention because failure to diagnose them early will produce serious disability in the form of a chronic painful wrist that is very unsatisfactory to treat.

A patient reports complaining of a sprained wrist from a fall on the outstretched hand. On examination there is a puffiness, perhaps distinct swelling in the "anatomical snuff box"; tenderness is present just below the radial styloid and limitation of wrist movements, especially dorsiflexion and radial deviation, is noted. Movements of the thumb and percussion of the head of the second metatarsal are painful. A good skiagram, carefully interpreted, will almost certainly reveal a fractured scaphoid. If under an anæsthetic mobility of the wrist is free, it should be immobilized on a long cock-up splint or in plaster for three weeks, the hand being in the position of grasp and the wrist in moderate dorsiflexion. After that period movements should be encouraged. If the wrist is still painful and not improving at the end of three months, excision of the scaphoid is indicated. Failing reduction of the fragments by manipulation, as evidenced by limited movements, especially dorsiflexion, or if there is much comminution of the bone, part or the whole of the scaphoid should be removed at once. Untreated, many of these fractures fail to unite and a painful chronic arthritis occurs. In this event the wrist should be maintained on a short cock-up splint for months. Should this fail, removal of the scaphoid or the half, usually the proximal, that appears to be giving trouble, may be undertaken, but the results of excision in late chronic arthritis are very uncertain. Excision in early cases gives a useful, fairly strong wrist with some radial deviation.

Dislocation of the semilunar, with or without fracture of the scaphoid, produces great swelling, pain and stiffness of the wirst and fingers. The pain may be intense, due to pressure on the median nerve. Anteriorly there is a distinct prominence under the flexor tendons, due to the forward dislocation of the bone. The absence of the latter from the first carpal row may be detected on palpation of the dorsal aspect of the wrist. The wrist cannot be dorsiflexed nor the fingers fully extended. The diagnosis should be made on this evidence. The lateral skiagram will confirm it, the semilunar standing out plainly in front of the carpus. In the antero-posterior view it may be missed, but here it will appear triangular in outline instead of the normal quadrilateral. Reduction should be attempted by hyperextension followed by acute flexion over a rounded rod or better still by the use of a Thomas wrench, the front bar maintaining extension by pressure over the semilunar while the hand is suddenly forcibly flexed. If reduction is not obtained within the first week, the bone should be excised without further delay. Excision gives excellent results. Untreated, great disability ensues. Apart from the mechanical obstruction to movements, sooner or later irritation of the median nerve arises. In late cases excision will always greatly improve function, although if arthritic changes are very pronounced, a painful wrist with limited mobility is all that can be expected.

A common complication of injuries caused by falls on the outstretched hand is a stiff shoulder. This is often not discovered until the patient begins to use the arm again. There are two conditions that cause this, one periarticular and the other intra-articular. The first is due to a slight tear of the insertion of the *supraspinatus* or capsule. There will be a history of pain and stiffness about the shoulder for the first two or three days after the injury, followed by limitation of movements when attempts are made to use the arm later. Treatment consists in active mobilization of the joint under anæsthesia followed by active use of the shoulder.

The other type is caused by a stub injury to the articular surface of the joint. An arthritis ensues, with pain of a boring, aching character coming on two or three weeks after the injury. Movements are limited in all directions and pain is increased by

[1] Read at a meeting of the South Australian Branch of the British Medical Association on August 30, 1928.

these attempts. The condition demands rest on an abduction splint with the local application of heat until the arthritis is quiescent; this requires several weeks. Then the joint is gradually mobilized by active movements and massage.

It is important to differentiate between these two types of stiff shoulder, as the treatment of the first is manipulation and movement and of the other rest.

### Lumbar Arthritis.

It was my privilege last year to hear a lecture by Professor Putti[1] entitled: "New Conceptions of the Pathogenesis of Sciatic Pain," and later to see some of his clinical material at Bologna. As the lecture has stimulated fresh interest in this subject, I thought a *résumé* of this work would be of interest.

Putti holds the view that an arthritis affecting one or more of the joints between the articular processes of the lumbar vertebræ is the cause of essential or idiopathic sciatica. Owing to the anatomical proximity of these joints to the nerve roots traversing the intervertebral canals, irritation of the roots ensues from direct pressure from distension of the joint, from congestion of the very rich plexus of veins that surrounds the roots, or by direct extension of inflammation from the joint capsule; again the sciatic pain may be purely a referred one.

It is necessary to reexamine the anatomy of these joints and the intervertebral foramina of the lumbar spine. The joints between the lumbar vertebræ lie in the sagittal plane, that between the fifth lumbar and first sacral in the coronal plane. The capsule of these joints forms part of the posterior wall of the intervertebral foramina. The foramen between the fifth lumbar and first sacral is the smallest, but transmits the largest root, the fifth lumbar root almost completely filling this canal. The foramen between the fourth and fifth lumbar is the next to smallest in size and transmits the next to largest root. The lumbo-sacral cord formed by these two roots forms the main part of the lumbo-sacral plexus. Certain abnormalities of the articular facets are common, particularly of the fifth lumbar vertebra. The lumbo-sacral articulation commonly has one joint in the sagittal and one in the coronal plane. This abnormality is also frequently seen throughout the lumbar spine, particularly the lower part of it.

These are admitted anatomical facts. The abnormalities tend (i) to alter the shape and size of the foramina, (ii) to alter the mechanics of this part of the spine, adding abnormal stresses and strains which tend to produce trauma of the joints, thus predisposing to or directly producing arthritis. The base of the spine is naturally subject to the heaviest work and always most liable to sprains. The actual arthritis may be traumatic or infective, the trauma in the latter case probably acting as a predisposing cause.

In such cases there is clinical evidence of a lumbar arthritis accompanying or preceding the sciatica. On examination there are limitation of movements of the lumbar spine, definite muscle spasm and tenderness over the joint affected. Scoliosis from muscle spasm is a common feature, in most cases contralateral to the sciatica, but sometimes homolateral and occasionally alternating. In addition we have the sciatic neuritis or neuralgia. In confirmation of the clinical diagnosis of lumbar arthritis, definite X ray evidence should be forthcoming. In acute lumbar arthritis there is an indefinite hazy outline to the joint. Chronic forms of arthritis are characterized by irregularity of joint outline or ankylosis of one or more joints with no joint space showing at all. There may be ankylosis of all the joints. Accompanying the above will usually be found abnormalities such as rotation of the facets previously mentioned. To obtain any reliable information "perfect radiograms and experienced examiners are indispensable." Antero-posterior, lateral and stereoscopic views are required, although I found that at Bologna Putti and his assistants were usually satisfied with antero-posterior and lateral views.

Treatment resolves itself into that for the arthritis. Rest is obtained in the early stage by recumbency and later by a removable plaster corset to immobilize the lumbar spine. Bier's hot air apparatus is used daily to provide hyperæmic treatment. The corset is worn for six to twelve months according to the progress and severity of the arthritis.

That is a brief outline of the views held by Putti which have had some further support in a recent excellent article by Feiling.[2] There are several points to which I would draw your attention. Lumbar arthritis as a cause of chronic lumbago, apart from sciatica, has not been stressed, yet for every patient with sciatica there must be several in whom the arthritis causes no more than a lumbago. Further, this arthritis affects isolated joints and is quite distinct from any of the classical types of *spondylitis deformans*. Feiling keenly advocates manipulation of the lumbar spine in chronic arthritis. This I have seen used with good effect at Liverpool. Great care must be taken, however, not to attempt this on any patient with *spondylitis deformans* of the ankylosing type or serious damage may be done.

In the past when examining skiagrams of the spine, we have been content, on seeing osteophytic lipping of the vertebral bodies, to diagnose osteoarthritis and to think that we have discovered the cause of the pain of which the patient complains. This belief received a well deserved blow from a recent investigation at the Mayo Clinic. The films of two thousand patients who had been examined for renal calculus, were reexamined for osteophytic outgrowths of the bodies. Of those over fifty years of age 70% manifested these changes and of this number only 20% had any symptoms at all referable to the spinal condition. In fact, lipping is evidence of a degenerative change that is usually symptomless, unless some trauma is superimposed. If my remarks do nothing more than draw your attention to the intraarticular joints in preference to the

edges of the vertebral bodies, they will not have been in vain.

### Osteoarthritis of the Hip.

Another condition frequently giving rise to sciatic pain is osteoarthritis of the hip. The common monarticular variety usually appears from middle age onwards and only occasionally in early life. The onset is usually subacute with pain on the inner side of the knee or down the sciatic, with stiffness of the hip joint. It is commonly diagnosed as sciatica at this stage, but if an examination of the hip joint be made, the classical signs of arthritis, muscle spasm, muscle wasting, limitation of movement in all directions, with commencing adduction and flexion deformity, will invariably be found. The diagnosis will be made early, if the condition is sought for and confirmatory skiagrams made.

Although this disease is progressive and produces great disability, the hopeless prognosis so commonly given is scarcely justified. With treatment instituted early and carefully maintained, most of the patients may be afforded great relief from pain and deformity, the progress of the disease delayed and in some cases brought to a standstill.

As in all other forms of arthritis, the basis of treatment in the acute stage is rest. If very active, a plaster spica is applied in abduction and extension and crutches provided. When the joint is quiescent and muscle spasm has subsided, fit a weight-relieving calliper and give daily stretching and abduction exercises. In less severe types a weight-relieving calliper can be fitted at once. Adduction deformity must be prevented, for once it arises a vicious circle is established, arthritis producing adduction and adduction increasing the arthritis, because of the additional strain placed on the joint at every step. If the active stage has passed and there is adduction deformity, daily passive stretching and active abduction exercises may overcome it. If not, the limb should be abducted under an anæsthetic and maintained there by a plaster spica until the joint settles down again after this trauma. Exercises should then be continued. Heat in one of the various forms should be applied in the active stages, diathermy being the best method. When the pain is worse on movement rather than on weight-bearing, manipulation under an anæsthetic will sometimes give definite relief with increased mobility. Raising the height of the boot on the affected side gives relief in the early stages, but as it increases adduction it must be used only temporarily. It may again be used in the later stages when adduction and flexion deformity are well developed.

Conservative means failing, the question of operative procedure must be considered if the general condition and age of the patient warrant it. The indications for surgical interference are: (i) persistent severe pain, (ii) increasing deformity in spite of treatment. Operation will almost certainly relieve the pain, but not necessarily the deformity. Of the various operations arthrodesis is that most commonly performed. Although a large percentage of the patients fails to get bony ankylosis, the fibrous ankylosis ensuing gives a painless hip but still usually with some deformity. In the monarticular type it is the operation which should be chosen.

The reconstruction operation such as Whitman's, with reforming of the neck and transplanting downwards of the trochanter, gives good results, if great attention can be given to after treatment, especially abduction exercises. It is, however, only suitable in a few selected cases in which the neck of the femur is reasonably healthy, with little absorption or shortening. In addition the patient must be keen to obtain a movable joint.

Jones's pseudarthrosis operation with excision of the neck and pegging of the trochanter over the head left in the acetabulum, is reserved for older people who cannot withstand the more severe operations necessitating disarticulation of the head and the longer restriction to bed entailed by such operations. It gives a movable and painless but unstable joint, requiring the wearing of a calliper. In bilateral cases a Whitman or a Jones is indicated.

All these operations have a place in the treatment. Needless to say, none is undertaken lightly, but in many cases the condition is so persistently painful and crippling that the patient is quite prepared to submit to any operative procedure promising relief of pain.

### Stabilizing Operation for Paralytic Deformities of the Foot.

After careful sustained postural treatment and muscle training for at least two years, in many cases of infantile paralysis we come to a stage at which the fullest possible recovery of muscles has been obtained. If this treatment has been conscientiously carried out, there should be no serious deformities. Unfortunately, for various reasons deformities have arisen or we have a foot unstable as to muscle balance with deformity prevented only by constant splinting. In mild deformities judicious tendon transplantation, after correction of the deformity, by manipulation and fixation in plaster may be sufficient, but has very limited use. A bone operation with or without tendon transplantation is necessary in the majority of instances to insure a permanent result. The stabilizing operation with arthrodesis of the mid-tarsal and subastragaloid joints is now generally accepted as the most suitable method. The principles are simple and sound. The ankle joint, except in a few instances, has no lateral movement. *Varus, valgus* and *calcaneus* deformities all occur at the mid-tarsal and subastragaloid joints. By fixation of these the posterior half of the foot is made rigid like an artificial one, lateral movements are abolished permanently, movement taking place only at the ankle joint in the sagittal plane. Any active inverting or everting muscles are transplanted, so that all muscles retaining any power act on the ankle joint. These principles are embodied in the Naughton-Dunn,[3] Hoke or what is commonly called the triple arthrodesis operation, that is fixation of the subastragaloid, calcaneo-cuboid and astragalo-scaphoid

joints. In both the Dunn and the Hoke operations in addition by removal of the scaphoid or neck of the astragalus the whole foot is moved back on the astragalus, a longer heel thus being made. This gives greater leverage to the calf muscles, so increasing their function, and greater stability to the foot.

The performance of the operation requires a good knowledge of the foot with very careful bone carpentry to insure that the wedges of bone removed from the joints are exactly sufficient to correct the deformity. The surgeon must see that the foot is put up in plaster in the best weight-bearing position with no lateral deviation. This is the "most important part of the operation," to quote Dunn. The position should be verified when the stitches are removed and a well fitting plaster applied and worn for four months. Weight-bearing is allowed at six weeks. There are two contraindications. The patient with weak quadriceps, requiring to wear a calliper permanently, needs no fixation, as this can be obtained by means of the splint and boot. The completely flail foot shows no tendency to become deformed and remains quite stable without a fixation operation.

By means of this operative procedure the child should walk without a splint and should have no further deformity. It is suitable and necessary to many old patients, allowing them to dispense with irons and cumbrous boots, the weight of which is an added burden to weak muscles, already overloaded.

It is frequently performed for other types of foot deformities, such as old congenital talipes, severe painful valgoid feet and fractures of the *os calcis* involving the subastragaloid joint.

The operation should not be performed before the age of eight. So successful has this operation proved that it has come into general use on both sides of the Atlantic within a few years. For further details of it I would refer you to Dunn's original communications.

#### References.

(1) V. Putti: "New Conceptions of the Pathogenesis of Sciatic Pain," *The Lancet*, July 9, 1927, page 53.
(2) A. Feiling: "Sciatica, Its Varieties and Treatment," *The British Medical Journal*, March 10, 1928, page 386.
(3) N. Dunn: *Journal of Orthopædic Surgery*, December, 1919, and "Stabilising Operations in the Treatment of Paralytic Deformities of the Foot," "Proceedings of the Royal Society of Medicine," 1921 to 1922, Volume XV, Section of Orthopædics, page 15.

## CARDIAC DYSPNŒA.[1]

By F. R. FRASER, M.D. (Edinburgh), F.R.C.P.,
*Professor of Medicine, University of London; Director of Medical Professional Clinic and Physician Saint Bartholomew's Hospital.*

DURING the nineteenth century clinical medicine was largely influenced by advances in morbid anatomy and by the recognition of the part played by bacteria in the causation of disease. During the past two or three decades advances in physiology have had a dominant influence on medical science. The increased knowledge of the functions of organs and of the ways in which the healthy body reacts to the ever-changing environment, has led to an increased knowledge of the ways in which these functions are disturbed by disease. The effect on clinical medicine has been to set us free from a too rigid classification of patients by recognized diagnostic labels and to enable us to pay more attention to the departures from the normal functioning displayed by the individual patient and to the therapeutic requirements of each. The approach of the medical man to the patient must always be through the symptoms of the patient and the advances in physiology must help the clinician to a clearer understanding of the causes of symptoms and of their indications for treatment. Clinical applications lag far behind advances in physiology, but the understanding of the types of glycosuria and the treatment of diabetes by "Insulin" and the relation of cardiac irregularities to prognosis and treatment are recent practical developments of physiological research. Much work on the physiology of respiration has been carried out in all parts of the world, perhaps preeminently at Oxford and Cambridge, but we are slow to apply the results of this work to the study of the respiratory disturbances seen in disease. No subject, however limited, can ever be completed and if a consideration of dyspnœa shows numerous and large gaps in our knowledge, these gaps will be more quickly filled by the recognition of their existence.

In considering the disturbances of respiration that result from disease, the respiratory apparatus may be regarded as consisting of the following structures: (*a*) The respiratory centre situated in the *medulla oblongata*, (*b*) the lungs, respiratory passages, walls of the thoracic cavity and the muscles of respiration forming the respiratory mechanism or bellows and (*c*) the blood and the circulatory apparatus.

The respiratory centre sends impulses to the muscles of respiration, which so alter the size of the thoracic cavity that the lungs expand and contract alternately and the air sacs within the lungs are kept supplied with air from the external atmosphere through the respiratory passages. By this means the blood circulating through the lungs continually takes up oxygen from the air sacs and being then distributed to the tissues of the body supplies these with the necessary oxygen for their needs and taking up carbon dioxide from the tissues carries it to the lungs, where it passes into the air sacs and so into the external atmosphere.

That the necessary amount of oxygen may be economically supplied to the tissues of the body under the varying conditions of normal life and the carbon dioxide efficiently removed, a close coordination between the respiratory centre and the blood flow is essential. Increased activity of the

[1] Being a lecture delivered at a special post-graduate course arranged by the Melbourne Permanent Committee for Post-graduate Work on August 8, 1928.

body requires increased oxygen supply and results in increased blood flow and increased ventilation of the air sacs. This increased ventilation is the result of increased activity of the respiratory centre.

In health the respiratory apparatus works efficiently without any consciousness on our part. The respiratory centre receives messages from the body and regulates the activity of the respiratory bellows according to the needs of the body. If the body needs more oxygen and has more carbon dioxide to eliminate, the centre sends the necessary messages to the muscles and the bellows act accordingly. In the constantly changing conditions of ordinary life the respiratory apparatus is constantly reacting and we are not conscious of the changes. If, however, the bellows cannot easily respond to the messages from the centre, we become conscious of the efforts and feel more or less distress. This is dyspnœa and it would seem, therefore, that there are two factors involved. The call on the respiration may be so great that healthy bellows cannot respond without effort that enters the consciousness. On the other hand the needs may be normal, but the apparatus so inefficient that the efforts to meet the demands produce consciousness of them. Or both the demands may be above the normal and the apparatus inefficient.

The consciousness may be affected apart from the effort necessary to meet the respiratory requirements, for the breath can be held until there is consciousness of the need for breathing. This may become so strong that there is discomfort and even an urgent desire for breathing that amounts to a dread of what will happen if breathing is not resumed.

Both forms of consciousness in relation to breathing, the consciousness of the movements and the consciousness of need, are described as shortness of breath or dyspnœa and constitute a subjective symptom which is found in many diseased states. Meakins[1] has attempted to define dyspnœa as "the conscious desire for increased pulmonary ventilation"; and Means[2] has said that "dyspnœa will occur whenever the respiratory mechanism cannot with ease functionate to the extent that bodily processes require." Both conceptions stress the subjective factor of consciousness and so involve the threshold of consciousness in the question of dyspnœa. The respiratory movements, however, are frequently so altered in disease that the onlooker knows that the patient has dyspnœa. Such a grade of respiratory disturbance as is obvious to the observer and is an objective evidence of disease, is a more suitable object for study than the lesser grades that are merely subjective and it is natural that most of the exact knowledge of dyspnœa is the result of observations on the severe grades. The question of the threshold of consciousness can thus be ignored and the causes of dyspnœa can be regarded as those factors which so disturb the mechanism of respiration that consciousness of effort may be produced.

### Causes of Dyspnœa.

Meakins and Davies[3] separated the causal factors into two groups: (i) Those that produce increased respiratory movements and increased pulmonary ventilation (or hyperpnœa) and (ii) those that limit the ability of the respiratory organs to meet the demands for increased pulmonary ventilation. This separation is valuable in a study of the causes of dyspnœa in disease.

The respiratory movements are controlled by the respiratory centre and J. S. Haldane[4] has discussed very fully in his book on respiration the factors that affect the centre to produce increased pulmonary ventilation. These factors may be divided into chemical and nervous. The chemical factors that are known to produce increased activity of the centre and hyperpnœa are (*a*) diminished oxygen pressure, (*b*) increased carbon dioxide pressure, (*c*) increased hydrogen ion concentration.

Whether increased breathing is the result of increased carbon dioxide in the arterial blood or of an increase of fixed acids or of oxygen lack may be determined by an examination of the arterial blood. In the first case there will be an increase in the content of the carbon dioxide in the blood and a high carbon dioxide pressure. When the stimulation is due to an increase of fixed acids, there will be diminished carbon dioxide content and a lowered carbon dioxide pressure through increased ventilation. In both cases the stimulus to the centre will be a raised hydrogen ion concentration and the arterial blood will be saturated with oxygen to the normal extent. When hyperpnœa is due to oxygen lack in the arterial blood and the resultant lowering of the threshold of the centre to carbon dioxide pressure and to hydrogen ion concentration, the arterial blood will show a lowered carbon dioxide content and pressure from increased ventilation and a lowered hydrogen ion concentration and will be saturated with oxygen to an extent less than normal.

If, however, there is increased breathing due to some stimulus at the centre other than an abnormality in the quality of the blood supplied to it, the examination of the arterial blood will show normal saturation with oxygen, low carbon dioxide content and pressure and low hydrogen ion concentration. These differences are shown in Table I.

On the examination of the arterial blood in cases of hyperpnœa single observations give results that occasionally indicate one of these conditions to be present. As a rule, however, this is not so, since the body cannot be considered as a respiratory system only. Changes in the reactions of the tissues generally and of the renal organs especially take place at the same time as the respiratory functions are disturbed or as a result of the disturbance. Henderson and Haggard[5] showed that in the presence of an altered carbon dioxide pressure in the blood a readjustment of base takes place between the tissues and the blood, so that the amount of alkali available for the carbon dioxide is altered. The alteration is in such a direction that

changes in the hydrogen ion concentration are prevented or are minimal. In the diseased states in which hyperpnœa occurs, more than one of the type conditions may be present; for example in pulmonary lesions both oxygen lack and raised carbon dioxide pressure may be present and kidney disturbances with faulty excretion of acid bodies may be present at the same time as pulmonary or circulatory abnormalities.

TABLE I.—CHANGES IN ARTERIAL BLOOD.

| Cause of Hyperpnœa | $O_2$ Saturation | $CO_2$ Volume | $CO_2$ Pressure | H-ion Concentration |
|---|---|---|---|---|
| Oxygen lack in arterial blood | Low | Low | Low | Low |
| Carbon dioxide excess in arterial blood | Normal | High | High | High |
| Fixed acids in excess .. | Normal | Low | Low | High |
| Stimulus at centre independent of quality of arterial blood | Normal | Low | Low | Low |

### Types of Cardiac Dyspnœa.

Among cardiac patients there are several distinctive forms of respiratory disturbances. Patients with mitral stenosis are short of breath to a degree that is seldom seen in other forms of heart disease till a much later stage in the story of failure. Patients with myocardial degeneration or aortic valve disease are seldom orthopnœic to the degree seen in patients with mitral stenosis until the terminal congestive form of failure arises. The cyanosis of a patient with mitral stenosis, who has many months or years of life to run, is comparable in degree to what is seen in the patient with arteriosclerosis and myocardial degeneration when much nearer the end. The cyanosis in mitral stenosis is of a much brighter colour than that of the patient dying with myocardial degeneration and when it does develope in the latter, it has a much more leaden tint.

The type of breathing in congestive failure, whether it appears early as in mitral stenosis or late as in myocardial degeneration, is shallow and rapid, quite unlike the breathing in uræmia or diabetic coma which is deep and slow. Periodic breathing of the Cheyne-Stokes type is rarely seen in congestive failure with its orthopnœa and shallow and rapid respiratory movements, but though rare in any type of case it is found in older patients with arterial disease before the late congestive failure sets in. Perhaps the most interesting form of all is that which occurs as a sudden paroxysm with increasing pulmonary œdema and failure. It most frequently occurs at night and may prove fatal.

In older patients there are so many possible factors in addition to the heart failure, such as arterial pulmonary and renal lesions, that it seemed advisable first to study the dyspnœa and orthopnœa seen in the comparatively younger patients with mitral stenosis and auricular fibrillation following rheumatism, since many cases are available in hospital and the respiratory disturbance is pronounced even when the patients are at rest in bed.

### Hyperpnœa.

In studying the cases of mitral stenosis for some cause for this increased breathing we[1] have examined the oxygen content of the arterial blood, that is of the blood supplied to the centre (Table II). With one exception they show no clear deficiency of oxygen. It should be noted that these patients are cyanosed even though there is no inefficiency in the oxygenation of the blood by the lungs. Other obvious possible causes of the hyperpnœa are increased carbon dioxide pressure and increased

TABLE II.—OXYGEN SATURATION OF ARTERIAL BLOOD IN CASES OF MITRAL STENOSIS AND AURICULAR FIBRILLATION WITH DYSPNŒA AND ORTHOPNŒA.

| Case No. | Sex | Age | Date | Ventricular Rate | Dyspnœa | Cyanosis | Oxygen Capacity, Vol. % | Oxygen Saturation, % |
|---|---|---|---|---|---|---|---|---|
| I | F. | 35 | 15.12.24 | 90 | + | + | 17·25 | 98 |
| II | M. | 25 | 22.12.24 | 116 | ++ | Slight | 17·7 | 97 |
| | | | 12. 1.25 | 70 | – | – | 16·9 | 97 |
| III | F. | 43 | 18. 6.23 | 104 | ++ | + | — | 96 |
| | | | 25. 6.23 | 80 | + | + | — | 95·3 |
| | | | 9. 7.23 | 52 | ++ | + | — | 97·5 |
| | | | 4. 1.25 | 150 | +++ | ++ | 16·4 | 98 |
| | | | 19. 1.25 | 76 | + | + | 17·5 | 98 |
| IV | F. | 27 | 9.11.25 | 60 | ++ | + | 18·4 | 97 |
| | | | 7.12.25 | 76 | + | + | 17·2 | 96 |
| | | | 13. 4.26 | 76 | ++ | + | 17·9 | 97 |
| V | M. | 34 | 25. 1.26 | 70 | ++ | + | 14·4 | 92 |
| | | | 8. 2.26 | 72 | ++ | + | 15·8 | 84 |
| VI | F. | 26 | 8.12.24 | 64 | Slight | Slight | 14·0 | 100 |
| VII | M. | 59 | 23. 4.23 | 78 | + | + | — | 94·4 |
| VIII | F. | 67 | 16. 6.24 | 76 | + | + | 21·2 | 93·8 |

hydrogen ion concentration. The next table (Table III) shows that there is no evidence in the arterial blood that these stimuli are present, indeed the carbon dioxide pressures are lower than normal and the blood more alkaline than normal, as if there was some stimulus at the centre causing increased

TABLE III.—CARBON DIOXIDE CONTENT AND PRESSURE AND HYDROGEN ION CONCENTRATION OF ARTERIAL BLOOD IN SAME CASES AS IN TABLE II.

| Case No. | Date | Dyspnœa | Arterial Blood | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | $CO_2$ Content | $CO_2$ Capacity at 40 mm. Hg. | $CO_2$ Pressure | | pH | |
| | | | | | From Dissociation Curve | From pH Pressure Curve | Colorimetric | Calculated |
| | | | Vol. % | Vol. % | mm. | mm. | | |
| I | 15.12.24 | + | 42·2 | 44 | 36 | 30 | 7·48 | 7·36 |
| II | 22.12.24 | ++ | 42·9 | 46 | 31 | 26 | 7·47 | 7·43 |
| | 12. 1.25 | – | 55·6 | 59 | 33 | 31 | 7·33 | 7·52 |
| III | 18. 6.23 | ++ | 55·7 | 44 | 26 | — | — | 7·44 |
| | 25. 6.23 | + | 40·2 | 43 | 38 | — | — | 7·34 |
| | 9. 7.23 | ++ | 41·4 | 46 | 32 | — | — | 7·41 |
| | 4. 1.25 | +++ | 33·6 | 45 | 21 | 17 | 7·46 | 7·50 |
| | 19. 1.25 | + | 43·4 | 50 | 29 | 29 | 7·34 | 7·47 |
| IV | 9.11.25 | ++ | 41·4 | 46 | 28 | 31 | 7·44 | 7·46 |
| | 7.12.25 | + | 43·2 | 47 | 32 | 31 | 7·37 | 7·42 |
| | 13. 4.26 | ++ | 39·9 | 42 | 35 | 35 | 7·36 | 7·34 |
| V | 15. 1.26 | ++ | 55·2 | 53 | 47 | 42 | 7·40 | 7·35 |
| | 8. 2.26 | ++ | 56·0 | 52 | 48 | 42 | 7·33 | 7·35 |
| VI | 8.12.24 | Slight | 51·4 | 48 | 45 | 42 | 7·38 | 7·33 |
| VII | 23. 4.23 | + | 40·0 | 38 | 43 | — | — | 7·26 |
| VIII | 16. 6.24 | + | 40·8 | 42 | 39 | — | — | 7·32 |

ventilation so that carbon dioxide is washed out and the alkalinity increased. In this group two cases appear to be exceptional. The patient of Case V

[1] The work has been continuous over a number of years and the following have taken part: G. Graham, C. F. Harris, D'Arcy Prendergast, R. Hilton and G. C. Linder.

had a low oxygen saturation and high carbon dioxide pressure. This patient had attacks of acute pain in the right side of the chest and friction was audible there. Case VII was exceptional in that the patient had a more acid reaction than normal. He was an old man with thickened arteries and granular casts in the urine.

On examining a series of patients with myocardial degeneration, heart failure and dyspnœa we find (Table IV and Table V) that it is only in the later or very severe degrees of failure that there is evidence of inefficient oxygenation of the blood in the lungs and retention of carbon dioxide, but that in many of the cases there is again evidence of over ventilation with a lowered carbon dioxide pressure and low hydrogen ion concentration. Further increased acidity and low carbon dioxide pressures are seen in patients near death and with failing renal functions. Cyanosis comparable to that seen in the cases with mitral stenosis is found in the cases with myocardial degeneration only when the failure is very advanced.

TABLE IV.—OXYGEN SATURATION OF ARTERIAL BLOOD IN CASES OF HIGH BLOOD-PRESSURE, ARTERIAL DISEASE AND MYOCARDIAL DEGENERATION, WITH DYSPNŒA AND ORTHOPNŒA.

| Case No. | Sex | Age | Date | Pulse Rate | Dyspnœa | Cyanosis | Oxygen Capacity, Vol. % | Oxygen Saturation, % |
|---|---|---|---|---|---|---|---|---|
| IX | M. | 60 | 28. 9.25 | 80 | + | — | 17·3 | 96 |
| | | | 31.11.25 | 108 | ++ | — | 13·8 | 96 |
| X | M. | 70 | 24.11.24 | 75 | + | — | 17·5 | 98 |
| | | | 1.12.24 | 100+ | + | + | 18·3 | 91 |
| XI | M. | 61 | 5.10.25 | 75 | + | — | 18·0 | 95 |
| XII | M. | 58 | 11. 2.24 | 64 | + | Slight | 16·8 | 100[1] |
| | | | 25. 2.24 | 75 | Periodic + Slightly periodic | — | 18·0 | 96[2] |
| | | | 17. 3.24 | 58 | Slight | — | 16·7 | 97·7 |
| XIII | F. | 53 | 28. 1.24 | 80 | +++ | ++ | 17·3 | 87·8 |
| | | | 3. 3.24 | 84 | ++ | + | 21·0 | 86·7 |
| | | | 31. 3.24 | 88 | + | Slight | 18·0 | 99 |
| XIV | F. | 48 | 21. 6.26 | 90 | + | + | 14·4 | 91 |

[1] Blood drawn during hyperpnœic phase.
[2] Blood drawn during both phase.

TABLE V.—CARBON DIOXIDE CONTENT AND PRESSURE AND HYDROGEN ION CONCENTRATION OF ARTERIAL BLOOD IN SAME CASES AS IN TABLE IV.

| Case No. | Date | Dyspnœa | Arterial Blood | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | $CO_2$ Content | $CO_2$ Capacity at 40 mm. Hg. | $CO_2$ Pressure: From Dissociation Curve | $CO_2$ Pressure: From pH Pressure Curve | pH: Colorimetric | pH: Calculated |
| | | | Vol. % | Vol.% | mm. | mm. | | |
| IX | 28. 9.25 | + | 43·7 | 49 | 31 | 29 | 7·38 | 7·44 |
| | 31.11.25 | ++ | 44·9 | 49 | 31 | 30 | 7·45 | 7·44 |
| X | 24.11.24 | + | 38·8 | 43 | 33 | 31 | 7·31 | 7·36 |
| | 1.12.24 | + | 27·6 | 31 | 27 | 27 | 7·34 | 7·29 |
| XI | 5.10.25 | + | 39·2 | 48 | 24 | 24 | 7·43 | 7·52 |
| XII | 11. 2.24 | + | 39·9 | 44 | 30 | — | — | 7·42 |
| | 25. 2.24 | Periodic + Slightly periodic | 37·1 | 43 | 29 | — | — | 7·41 |
| | 17. 3.24 | Slight | 38·8 | 45 | 28 | — | — | 7·44 |
| XIII | 28. 1.24 | +++ | 45·1 | 42 | 48 | — | — | 7·26 |
| | 3. 3.24 | ++ | 51·8 | 46 | 51 | — | — | 7·31 |
| | 31. 3.24 | + | 44·0 | 46 | 36 | — | — | 7·39 |
| XIV | 21. 6.26 | + | 39·0 | 43 | 32 | 35 | 7·37 | 7·36 |

In a small series of patients with pulmonary disease in addition to heart failure (Table VI and Table VII), however, carbon dioxide retention is more general, with in some cases inefficient oxygenation in the lungs as well, but in the recovered stages the same tendency to low carbon dioxide pressure and reduced acidity is found.

TABLE VI.—OXYGEN SATURATION OF ARTERIAL BLOOD IN THREE CASES OF CHRONIC PULMONARY DISEASE WITH HEART FAILURE AND IN TWO CASES OF HEART FAILURE NOT INCLUDED IN OTHER GROUPS. ALL HAD DYSPNŒA AND ORTHOPNŒA.

| Case No. | Sex | Age | Date | Pulse Rate | Dyspnœa | Cyanosis | Oxygen Capacity, Vol. % | Oxygen Saturation, % |
|---|---|---|---|---|---|---|---|---|
| XV | M. | 51 | 15. 6.25 | 75 | ++ | Slight | 19·5 | 100 |
| | | | 29. 6.25 | 75 | + | Slight | 19·75 | 100 |
| | | | 15. 2.26 | 96 | +++ | + | 19·3 | 100 |
| | | | 22. 2.26 | 84 | + | Slight | 19·8 | 96 |
| XVI | M. | 59 | 7. 6.26 | 80 | + | Slight | 17·5 | 94 |
| XVII | M. | 35 | 19.10.25 | 80 | ++ | ++ | 19·4 | 91 |
| XVIII | M. | 56 | 18. 1.26 | 92 | + *Angina pectoris* | Slight | 16·3 | 96 |
| XIX | F. | — | 12.10.25 | — | Slight | — | 14·3 | 92 |

TABLE VII.—CARBON DIOXIDE CONTENT AND PRESSURE AND HYDROGEN ION CONCENTRATION OF ARTERIAL BLOOD IN SAME CASES AS IN TABLE VI.

| Case No. | Date | Dyspnœa | Arterial Blood | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | $CO_2$ Content | $CO_2$ Capacity at 40 mm. Hg. | $CO_2$ Pressure: From Dissociation Curve | $CO_2$ Pressure: From pH Pressure Curve | pH: Colorimetric | pH: Calculated |
| | | | Vol. % | Vol. % | mm. | mm. | | |
| XV | 15. 6.25 | ++ | 57·8 | — | — | — | — | — |
| | 29. 6.25 | + | 49·6 | 53 | 33 | 28 | 7·33 | 7·48 |
| | 15. 2.26 | +++ | 47·8 | 44 | 54[1] | 40 | 7·42 | 7·23[1] |
| | 22. 2.26 | + | 50·5 | 47 | 48 | 41 | 7·40 | 7·27 |
| XVI | 7. 6.26 | + | 52·4 | 48 | 50 | — | 7·42 | 7·30 |
| XVII | 19.10.25 | ++ | 66·9 | 59 | 70[1] | — | 7·37 | 7·26[1] |
| XVIII | 18. 1.26 | + | 47·3 | 48 | 39 | 34 | 7·35 | 7·37 |
| XIX | 12.10.25 | Slight | 51·4 | 51 | 41 | 39 | 7·42 | 7·38 |

[1] Figures uncertain since that for carbon dioxide pressure was obtained by extrapolation.

It seems justifiable to conclude from these observations that there is in general in the dyspnœa of heart failure some stimulus at the centre independent of the quality of the arterial blood causing increased breathing with washing out of carbon dioxide and reduced acidity in the arterial blood, but that in the presence of severe pulmonary lesions, either independent of the heart failure or due to advanced heart failure, there is a retention of carbon dioxide or a gaseous acidosis, while in patients near death or in very severe failure there may be in addition an increased acidity and a reduced carbon dioxide tension such as would result from the faulty elimination of non-volatile acid bodies (see Figure I).

In most forms of heart failure the essential disturbance is believed to be a reduction in the amount of blood circulating in a given time. The methods available for the estimation of the minute-volume are not satisfactory, but enough work has been done to support the clinical evidence and to show that in heart failure with congestion and stasis there is a diminished amount of blood passing through the tissues in a given time. The blood thus takes longer to pass through the tissues and

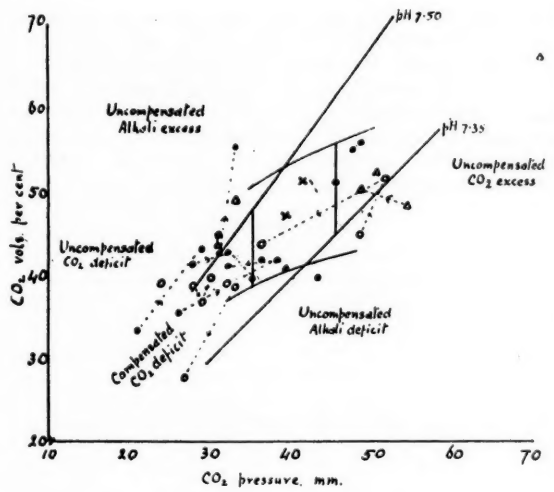


FIGURE I.

in its passage there will be more oxygen extracted from it and the mean pressure of oxygen to which the tissues are exposed will thus be reduced. This is a form of anoxæmia independent of the character of the arterial blood as it leaves the lungs.

This anoxæmia of the tissues will be present at the respiratory centre and is a sufficient stimulus to cause the increased ventilation. At the same time the blood must take up more carbon dioxide and the mean pressure of carbon dioxide in the tissues will be increased. This will be an additional stimulus, but the over-ventilation that results from the oxygen lack washes out an excess of carbon dioxide from the lungs and so counteracts this carbon dioxide effect.

If carbon dioxide retention were a constant factor in cardiac dyspnœa, one would expect from the observations of Henderson and Haggard[5] that the carbon dioxide capacities would be high, but many of the patients in this series have low capacities in the absence of renal disease and in the less severe stages of failure. It is unlikely, therefore, that in them there is a raised carbon dioxide pressure in the tissues.

Peabody, Meyer and Du Bois[6] have estimated the metabolism in cases of cardiac insufficiency with dyspnœa and find that it is increased over the normal and that the increase may be as great as 25% to 50%. Their results have been confirmed by Hamburger and Lev[7] and by Shapiro.[8] Eppinger, Papp and Schwarz[9] estimated the difference in the oxygen content of the arterial blood and of the blood from the arm veins in normal subjects and in cases of different types of dyspnœa. In cases of heart failure with valvular disease they found a pronounced increase in the amount of oxygen extracted by the tissues from a given volume of blood in the majority of cases. This increased utilization was more evident in cases of mitral valve disease than in cases of aortic valve disease. They point out that blood from the arm veins does not necessarily give a true indication of the general circulation. In cases of dyspnœa with high blood pressure and heart failure they did not find evidence of increased utilization.

I may thus sum up the cause of the increased breathing or hyperpnœa in heart failure to be due to anoxæmia due to stasis, but that if pulmonary disease is present or if the secondary changes in the lungs, such as œdema, are severe, there will be an additional stimulus in carbon dioxide excess and that near death or if renal functions are severely disturbed, there is an additional factor in the retention of non-volatile acid bodies and a raised hydrogen ion concentration at the centre.

In accounting for dyspnœa it is not sufficient, however, to show that there is increased ventilation, unless we can show that this is so great that the pulmonary apparatus cannot meet the demand easily or that there is also a diminution in the capacity of the bellows and their mechanism.

### Vital Capacity.

In quiet, healthy breathing the amount of air that is inspired and expired at each respiratory cycle is about 500 cubic centimetres. This is called the tidal air. The amount that can be forcibly expired after a forced inspiration is between four and five litres. This is called the vital capacity and is divided into the complemental air which is the amount that can be forcibly inspired from the mid-position and the reserve air or the amount that can be forcibly expired from the mid-position. After a forcible expiration there is still a considerable volume of air in the lungs, called the residual air. The tidal air can be greatly increased without dyspnœa arising and Peabody[10] showed that if hyperpnœa is induced by breathing increasing concentrations of carbon dioxide, the tidal air may be increased 400% before distress becomes urgent. The actual increase before the onset of distress varies considerably with individuals. Means and Balboni[11] showed that if the vital capacity is reduced by half in artificial pneumothorax, the tidal air increases with increasing concentrations of carbon dioxide as in the normal, but that dyspnœa appears much earlier. It is evident, then, that the relation of tidal air to vital capacity is important in determining the onset of dyspnœa. Vital capacities in healthy individuals have been carefully studied and Dreyer's conclusion[12] that it is a simple function of the area of the body surface is generally accepted. The vital capacity in cases of congestive heart failure has been studied by Siebeck,[13] by Bittorf and Forschbach[14] and by Peabody and Wentworth.[15] They find a very constant reduction in vital capacity which may be as low as one litre. Further, Peabody and Wentworth followed the condition in patients from stages of severe failure to those of comparative recovery and found that as recovery occurred, the vital capacity rose. The reduction of vital capacity affects the reserve air more than the complemental. To ascertain the cause of the reduced vital capacity, Drinker, Peabody and Blumgart[16] carried out

animal experiments. They concluded that the loss of elasticity of the lungs that results from congestion, is the main factor and that the greater the pressure in the pulmonary circuit, the less the vital capacity. They point out that the actual enlargement of the heart and the increased quantity of blood in the pulmonary vessels must reduce somewhat the air-containing space in the thorax. The onset of œdema of the lungs and of fluid in the pleural cavities, as extreme failure sets in, will still further reduce the vital capacity. That a reduction in vital capacity is an essential factor in the production of cardiac dyspnœa is supported by the examination of the arterial blood in two patients with active heart disease who were without any respiratory distress when lying flat in bed. A patient (Case XX), aged thirty-eight, had subacute bacterial endocarditis with aortic regurgitation and mitral valve disease. The heart was dilated and there was œdema of the legs and a few râles at the bases of the lungs. Another patient (Case XXI) had active rheumatic carditis with aortic regurgitation and mitral disease and the heart was dilated. The results of the examination in these cases are shown in Table VIII. The normal oxygen saturations, low carbon dioxide pressures and volumes with low normal capacities and values for the calculated hydrogen ion concentration in the alkaline half of the normal range are similar to those found in the younger patients with mitral stenosis and auricular fibrillation and severe dyspnœa. The character of the arterial blood indicates increased ventilation, therefore, in both the dyspnœic patients and in the patients of Cases XX and XXI. The patients with dyspnœa differed from those two others, apart from the presence of respiratory distress, in that they had a high colour and more or less cyanosis indicative of some degree of venous engorgement. It is probable, therefore, that the incidence of the dyspnœa was determined by the reduction of vital capacity due to pulmonary vascular engorgement. The mechanism of heart failure in cases of mitral stenosis differs from that in cases of high blood pressure and myocardial degeneration in that pulmonary engorgement occurs at a much earlier stage in the failure and this has most likely a causal relationship with the early incidence of dyspnœa in mitral stenosis and its later incidence in cases of myocardial degeneration and high blood pressure when the stage of advanced failure is reached. It would appear, therefore, that the effort necessary to effect the required ventilation in the presence of the reduced vital capacity is a factor in bringing the respiratory act into the field of consciousness and producing distress.

Table VIII.—Result of Examination of Arterial Blood in Two Cases of Acute Valvular Disease without Evidence of Dyspnœa or Heart Failure at Rest.

| Case No. | Age | Date | Dyspnœa at Rest | Arterial Blood | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | $O_2$ Capacity | $O_2$ Saturation | $CO_2$ Content | $CO_2$ Capacity at 40 mm. Hg. | $CO_2$ Pressure from Dissociation Curve | pH Calculated |
| | | | | Vol. % | % | Vol. % | Vol. % | mm. | |
| 20 | 38 | 30.6.24 | Nil | 17·7 | 100 | 36·7 | 42 | 28 | 7·42 |
| 21 | 16 | 14.4.24 | Nil | 14·8 | 97 | 40·0 | 43 | 29 | 7·44 |

### Shallow Breathing.

The type of breathing in cardiac dyspnœa is rapid and shallow as a rule. The chemical stimuli at the centre regulate the amount of alveolar ventilation and when it is considered that the presence of a considerable dead space in the upper respiratory passages and bronchial tree must result in waste each time this space is filled and emptied, it is obvious that the deeper and fewer the respiratory movements are, the more economical they will be in establishing the required ventilation. The type of breathing is normally regulated by the Hering-Breuer reflex. Afferent stimuli from the lungs are conveyed to the centre by the vagus nerves, so that when inspiration has proceeded to a certain extent, the inspiratory movement is inhibited and expiration takes place. Means[2] considers that the respiratory centre and the vagus inhibition are so regulated in health that a happy medium exists between slow deep breathing and rapid shallow breathing. Slower and deeper breathing would involve active respiratory efforts to expel some of the reserve air and so lead to greater exertion than is necessary when normal tidal respiration with its passive expiratory phase is occurring, while shallower respiration by increasing the importance of the dead space would cause more rapid breathing and augment the work of the respiratory musculature in effecting the necessary alveolar ventilation. It is conceivable that some exaggeration of the vagus inhibition relative to the activity of the respiratory centre can bring about the shallow rapid form of breathing seen in cardiac dyspnœa and Haldane believes that the reflex is exaggerated through some stimulus from the heart or elsewhere so that the inspiratory movement is more limited than in health. Haldane, Meakins and Priestley[17] showed that shallow and correspondingly frequent breathing resulted from long continued oxygen want and considered it due to fatigue of the centre. Davies, Haldane and Priestley[18] showed that this type of breathing resulted as a response to excessive and prolonged resistance. The rapid and shallow character of the breathing in cardiac dyspnœa may be the result, then, of a relative exaggeration of the vagus inhibition of the inspiratory act, but the appearance of this character comparatively early in the course of mitral stenosis and the experimental work of Peabody point to the mechanical limitation of expansion and reduction of vital capacity consequent on the vascular engorgement as an undoubted factor. In the later stages of failure gross œdema of the lungs will still further limit the depth of breathing and the fatigue of the centre due to anoxæmia and increased resistance must add

its effect. That shallow breathing can cause anoxæmia has been demonstrated by Haldane, Meakins and Priestley.[17] A vicious circle will thus be established and a dangerous and urgent stage of failure will be reached. The rapidity of the breathing that accompanies the shallowness in order to effect the necessary ventilation is probably a factor in causing consciousness of the respiratory movements and distress.

### Orthopnœa.

The cause of orthopnœa has been investigated by a number of workers and it has been shown that the vital capacity is reduced when the posture is altered from the upright to the prone position. When the vital capacity is already reduced by the circulatory disturbance, it is easy to understand why the patient with congestive failure prefers the orthopnœic position. Haldane and his colleagues have further shown that when the breathing is of the shallow, rapid type, anoxæmia is more readily produced when the subject is lying down.

### Periodic Breathing.

As the result of the work of Pembrey and Allen[19] and of Douglas and Haldane[20] it is generally conceded that periodic breathing of the Cheyne-Stokes type is the result of oxygen lack at the centre. In the patients with arterio-sclerosis and myocardial degeneration, which are the group in which this type of breathing is seen most often, it is not difficult to understand how the respiratory centre should be in a state of oxygen want, but there is difficulty in understanding why it is not seen in congestive failure in which also the centre is suffering from oxygen lack, and indeed why it is a rare condition in all classes of failure. Poulton and Haldane[21] showed that as the result of oxygen lack not only does the centre react about a lower threshold of carbon dioxide pressure, but that it is much more sensitive to changes in this pressure. This increased sensitiveness of the centre is essential to the occurrence of periodic breathing. For periodic breathing to occur, therefore, it would seem necessary that there should be not only oxygen lack at the centre, but also that the respiratory apparatus should be able to respond to the call of the centre by considerable increase in the ventilation, so that the carbon dioxide is rapidly washed out and its pressure lowered, so that apnœa results. In the congestive forms of failure, such as in mitral stenosis and the later stages of myocardial degeneration, the vital capacity is reduced and the lungs cannot respond by a large increase in ventilation and the washing out of carbon dioxide and so in failure of this type periodic breathing is rarely seen.

So far, then, we can account for the hyperpnœa and dyspnœa, the shallow breathing, the orthopnœa and the rarity of periodic breathing by the slow circulation and the reduced vital capacity. Both the slowing of the circulation and the reduction in the vital capacity are the consequence of myocardial inefficiency and the essential treatment for these conditions must be treatment for the myocardial inefficiency, rest, digitalis, bleeding and so forth. On the question of the use of oxygen and the indications for its use there is also evidence. The bright cyanosis of the patient with mitral stenosis is due not to inefficient aeration of the blood in the lungs but to stasis and oxygen administration cannot affect this. Cyanosis, however, in the patients with pulmonary disease as well as heart failure or with arterial disease and myocardial degeneration, is associated with inefficient oxygenation in the lungs and in such oxygen administration is indicated.

### Paroxysmal Dyspnœa.

There is another form of cardiac dyspnœa which I have not yet discussed, paroxysmal dyspnœa or cardiac asthma, and consideration of this form is important, as it raises the question of the use of morphine in cardiac dyspnœa. In such attacks there is general distress and violent respiratory movements. The patient becomes more and more cyanosed and if the attack continues, œdema of the lungs developes. The patient may die in the attack. Morphine terminates the attack and after its use the violence of the respiratory movements diminishes, the cyanosis clears up and the râles gradually diminish. Eppinger, Papp and Schwarz,[9] using a number of methods of investigation, came to the conclusion that in paroxysmal cardiac dyspnœa the minute-volume of the circulation is higher during the attack and falls when the attack terminates. This conclusion is at first sight unexpected. Various causes have been suggested for these paroxysms. They commonly occur in the night and the patient is usually suffering from heart failure with severe myocardial degeneration and raised blood pressure. It may be that the more recumbent position during sleep allows of congestion and œdema gradually to accumulate until the patient is roused by the call for increased ventilation.

Spasm of the cerebral vessels with increased oxygen want at the centre, spasm of the bronchial muscles or some disablement of the efficiency of the left ventricle, such as coronary thrombosis or a new rhythm, have all been suggested as the cause of onset. Whatever the onset is due to, the results of Eppinger, Papp and Schwarz can be explained by the violent respiratory movements causing a pumping of blood to the heart from the veins so that the right ventricle pumps to the pulmonary circulation and so to the left side more than the already disabled and strained left ventricle can handle. Increasing pulmonary congestion and œdema will thus result and a circulation rate as great as the left ventricle is capable of. If the attack continues, the left ventricle will fail. Morphine by depressing the respiratory centre will quiet the respiratory efforts and the pumping action and so give the left ventricular muscle a chance to keep up with the demands made on it by the right ventricle; the attack will, therefore, subside and the pulmonary congestion and œdema diminish.

If this explanation be the correct one, it would appear that dyspnœa is not always a beneficial effort of the body to compensate, but that it may be a dangerous reaction and one that should be directly checked. To depress the respiratory centre by morphine is running the risk of so lowering its threshold for the physiological stimuli that dangerous anoxæmia will result, but it is possible that this risk must be taken to save the heart from complete failure. If the centre is in a state of fatigue and the respiratory efforts shallow and feeble, it is probable that the risk is too great and the giving of morphine will result in respiratory failure. If, however, the respiratory efforts are violent and the lungs œdematous and not expanding in measure with the efforts so that there is a sucking action with each inspiration, morphine is of great benefit. It should be combined with oxygen administration to counteract, if possible, the anoxæmia that may result from its use (See Table IX). The value of

Table IX.—Effect of Morphine on the Dyspnœa and Characters of the Arterial Blood in Case No. XV.

| Date | Morphine | Dyspnœa | Arterial Blood | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | $CO_2$ Pressure | | pH | |
| | | | $O_2$ Saturation | $CO_2$ Content | From Dissociation Curve | From pH Pressure Curve | Colorimetric | Calculated |
| | | | % | Vol. % | mm. | mm. | | |
| 15.2.26 | Before | +++ | 100 | 47·8 | 54[1] | 40 | 7·42 | 7·23[1] |
| | ½ hour after gr. ½ | — | 80 | 50·8 | 60 | 56 | 7·37 | 7·20 |
| 22.2.26 | Before | + | 96 | 50·5 | 48 | 41 | 7·40 | 7·27 |
| | ½ hour after gr. ½ | Slight | 95 | 52·8 | 50 | 45 | 7·42 | 7·27 |

[1] Figures uncertain since that for carbon dioxide pressure was obtained by extrapolation.

morphine in paroxysmal dyspnœa is undoubted and it is probable that it should be used more than it is in other types of cardiac dyspnœa. It must at present be used with great judgement, as our methods of administering oxygen to severely dyspnœic patients are very unsatisfactory. Improved methods of administering oxygen would allow of more latitude in the use of morphine and enable a very distressing symptom to be relieved without danger to the patient.

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## ULNAR PARALYSIS: A NEW OPERATION.


By Lockyer Potter, M.D. (Melbourne),
*Melbourne.*


In the writer's opinion the results of the treatment of ulnar paralysis during the war period were not good in regard to recovery of function of the intrinsic muscles of the hand supplied by that nerve. This opinion is based on an experience of fifty cases. Some were personally treated by operation and otherwise, many had been operated on by others and all were subjected to prolonged observation.

The work was carried out in close association with Dr. Paul Dane, of Melbourne, who published the results of his own very careful studies some years ago,(1) but Dr. Dane is not identified necessarily with the opinions expressed by the present writer.

In all cases the nerve had been divided and subsequently sutured. In no case did these intrinsic muscles regain sufficient power to overcome the deformity or to produce a useful degree of movement

or indeed in the great majority if not in all to produce any movement; nor was there any discernible increase in bulk, but the wasting persisted. The slips of the *flexor profundus digitorum* supplied by the ulnar nerve showed an equally unsatisfactory degree of recovery, but the *flexor carpi ulnaris* regained good voluntary function in all instances.

Therefore doubt arose in the mind as to whether there was anything to be gained by subjecting the patients to a long course of post-operative physical treatment after the nerve had been sutured and the wound had healed.

Since there was no evidence that the muscles had benefited and therefore that the motor fibres had been aided in their regeneration, it would appear that the regeneration of the sensory fibres also had not been favourably influenced by physical treatment. Indeed, the impulse to grow is so enormous in all living tissues and particularly so in nerve fibres, that it is difficult to imagine in what way regeneration could be assisted after careful suture had been performed.

Was not this long period of tedious post-operative physical treatment, running into many months and years of daily attendance, merely a useless, costly procedure involving waste of time and energy?

In these circumstances an operation was devised by the writer and carried out in a limited number of cases in the early post-war period. It is based on certain principles referred to in earlier contributions[2] and its reason lies in the unsatisfactory degree of recovery to be expected with consequent loss of power and deformity.

In ulnar paralysis the muscle slips of the *flexor profundus digitorum* to the ring and little fingers are involved, together with the two ulnar lumbricals, the interossei, the deep head of the *flexor brevis pollicis*, the *adductor obliquus pollicis* and the *adductor transversus pollicis*. As a result of the paralysis a passive hyperextension of the two medial (ulnar or inner) fingers occurs at their metacarpo-phalangeal articulations with flexion at the interdigital joints, owing to the corresponding lumbricals being affected (see Figure I). Well marked as this

Figure I.
This shows characteristic wasting and deformity in a case of ulnar paralysis. The hand is at rest.

condition is when the hand is at rest, it becomes an actual clawing when extension of the fingers is attempted (see Figure II) because, apart from

Figure II.
Palmar view of the hand in a typical case of ulnar paralysis: attempted extension of fingers.

the now purposeful action of the long extensor increasing the hyperextension referred to, the *flexor sublimis digitorum* comes strongly into action and plays an important part in producing the characteristic deformity. For extension of the two distal phalanges to occur there must be fixation at the corresponding metacarpo-phalangeal articulation which in its turn calls for equal and synchronous action of the muscles acting on that articulation; but on account of the paralysis of the lumbricals such action is unbalanced and the *flexor sublimis digitorum*, coming into action primarily to help to fix the metacarpo-phalangeal joint, overcomes at the proximal interdigital joint the *extensor communis digitorum*, the action of which now is not aided by a sound lumbrical; it causes flexion there and continuing its action, produces a still further increase of hyperextension at the metacarpo-phalangeal articulation. The action of the long extensor on the distal interphalangeal joint is so slight that it cannot overcome any feeble contraction that may be present in the *flexor profundus digitorum* nor in the case of no recovery having occurred in the latter can it overcome the stiffness that developed as the joint gravitated into flexion.

How can that hyperextension be overcome? The powerful extensors cannot overcome it, however great may be the efforts of so-called reeducation. They cannot overcome it because, powerful as they are, they do not act directly on the joint and because they function at poor mechanical advantage. The slender little lumbricals have this great power on account of the mechanical advantage with which

they act. It must be remembered, however, that in ulnar paralysis not only are these fragile little muscles paralysed, but the base from which they act, has been rendered insecure by the same paralysis, because the tendons which give them their origin, the two inner (or medial) deep flexors, lie slack. The deformity of the third or ring finger is less pronounced because the corresponding lumbrical obtains its origin partly from the deep flexor tendon of the middle finger, which latter corresponds to a part of the *flexor profundus digitorum* that is not paralysed, being supplied by the median nerve. We recognize, therefore, that, as a result of the paralysis of the lumbricals concerned, the stability of the metacarpo-phalangeal joints has been impaired and that such stability has been further affected because the paralysed muscles cannot act even as simple slings, since their own essential stability has been lessened by the slackness of the corresponding deep tendons.

If an attempt be made to extend the affected fingers, the proximal phalanges assume a more extreme position of hyperextension and the distal phalanges remain flexed (see Figure IIIA).

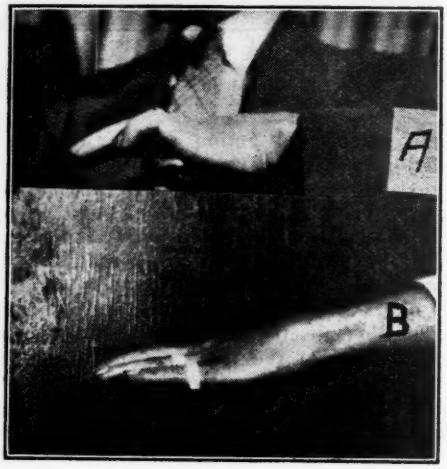


FIGURE III.
A demonstrates increased hyperextension of the proximal phalanges of the ring and little fingers, produced by attempted extension at the inter-phalangeal joints. B shows hyperextension prevented in the same person by a loop of strapping passing across the dorsum of the proximal segment of the ring and little fingers and around their sides and thence proximally to be attached to the skin of the palm. The action of the lumbricals is thus imitated.

But if the metacarpo-phalangeal joints be fixed by a loop of strapping (see Figure IIIB) or by an appropriate grip (see Figure IV), the fingers may be extended without the production of deformity.

The disability is now understood. The two distal phalanges cannot be extended because the proximal phalanx is unstable; there being no secure base, effective movement cannot occur. Therefore, should any measure be contemplated in order to eliminate the deformity associated with ulnar paralysis, attention must be directed to the stabilization of the metacarpo-phalangeal joints concerned.

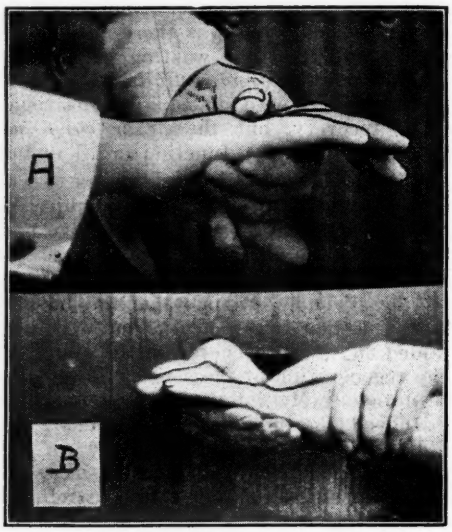


FIGURE IV.
The figure illustrates fixation by an appropriate grip of the metacarpo-phalangeal joints of the affected fingers which allows of extension occurring at the inter-phalangeal joints. A shows this fixation in a sound hand. B shows it in a hand affected by ulnar paralysis.

The object of the operation to be described is primarily the stabilization of the appropriate metacarpo-phalangeal joints by opposing the action of the long extensor and of the *flexor sublimis digitorum*. This is done by dividing transversely the tendon of the paralysed deep flexor as it traverses the osteo-fibrous canal of the finger; the proximal part of the divided tendon is transplanted dorsally and is used to fix the metacarpo-phalangeal articulation. An attempt is made to secure effective movement of the distal phalanx by transplanting dorsally the distal part of the same tendon.

In Figure V is seen the manner in which the tendon of the paralysed *flexor profundus digitorum* is made use of. Each of the four fingers has been dissected in order to illustrate the various stages of the operation which, of course, applies to the ring and little fingers only.

At A in Figure V the little finger is seen opened up on its ventral surface and the digital tendon sheath exposed. The sheath has been cut away in three situations indicated by the numerals 1, 2, 3 and corresponding to the neighbourhood of the joints of the finger. The numeral 1 corresponds to a point just proximal to the distal interphalangeal joint; exposed in the opening of the sheath is seen the insertion of the *flexor profundus digitorum*. Numeral 2 indicates an opening in the tendon sheath just proximal to the proximal interphalangeal joint; the two tendons are exposed, the deep tendon lying in the middle, the diverging slips of the superficial tendon lying on either side and retracted. Numeral 3 indicates the third opening in the digital sheath; the superficial tendon is retracted medially and the deep tendon laterally. In carrying out the operation the paralysed tendon is divided at 2 and the distal and

proximal ends of the divided tendon withdrawn from the sheath at 1 and 3 respectively.

At B the proximal and distal parts of the cut tendon are withdrawn at the points referred to, the distal end being thrown distally along the distal phalanx of the ring finger.

At C the proximal end of the cut tendon is divided longitudinally and the medial of the two divisions so formed is carried through the digital tendon sheath to its appropriate side of the finger. The distal end also of the tendon has been divided longitudinally and the two divisions again divided similarly in order to obtain increased length.

FIGURE V.

A First Stage.

Digital canal opened to show tendons 1, 2 and 3.

1 Insertion of *flexor profundus digitorum.*
2 Diverging slips of tendon of *flexor sublimis digitorum* retracted to either side of tendon of *flexor profundus digitorum*, the latter being seen in the middle.
3 Superficial tendon retracted medially, that is ulna-wards and deep tendon laterally.

B Second Stage.

The deep tendon has been transversely divided near the proximal interphalangeal joint and the proximal and distal cut ends withdrawn through openings in the digital sheath near the metacarpo-phalangeal and distal interphalangeal joints respectively.

4 Distal cut ends of deep tendon.
5 Proximal cut end of deep tendon withdrawn to the radial (outer) side of the superficial tendon.

C Third Stage.

The withdrawn distal end of the transversely cut tendon has been longitudinally divided and the divisions further subdivided longitudinally. The withdrawn proximal end also has been longitudinally divided and its ulnar division passed beneath the superficial tendon to emerge on the ulnar side of that tendon.

6-6 Divisions of distal end of cut tendon.
7-7 Divisions of proximal end of cut tendon.

D Fourth Stage.

The divided proximal and distal ends of the cut tendon are seen passing dorsally and encircling the proximal and middle phalanges respectively.

8-8 Distal subdivisions.
9-9 Proximal ditto.

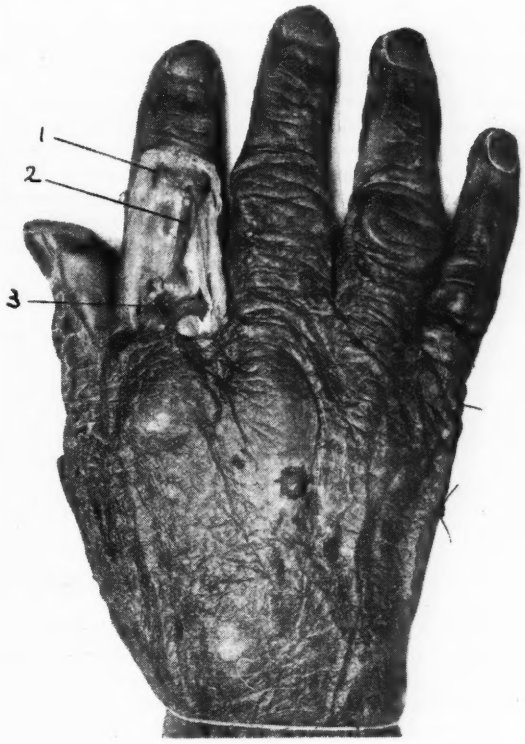


FIGURE VI.

Divisions of the tendons as they appear on the dorsal aspect of the finger.

1. The distal subdivisions sutured together over the dorsal aspect of the middle phalanx.
2. The same subdivisions carried across the proximal interphalangeal joints and sutured to the tendon of the long extensor.
3. Proximal subdivisions sutured together across the dorsal aspect of the proximal phalanx.

At D it is seen that each of the two portions of tissue obtained by splitting the proximal part of the divided tendon has been carried around the corresponding side of the proximal phalanx, passing through the soft tissue next to the bone. The split distal end of the tendon has been similarly made to encircle the middle phalanx.

In this dissection of the index finger the whole of the digital tendon sheath has been removed in order to show more clearly the divisions of the tendon leaving the palmar surface of the finger. Incidentally the tendon of the *flexor sublimis digitorum* is seen lying on the floor of its osteo-fibrous canal and its natural divisions passing distally to their insertions.

Figure VI is a dorsal view of the hand showing the manner in which the transplanted extremities of the divided tendon are fixed in their new situations. Proximally are seen the divisions of the proximal part of the tendon sutured together over the dorsal aspect of the middle of the proximal

phalanx. Distally the divisions of the split distal end of the paralysed tendon have been approximated and sutured together over the middle phalanx and the remainder of the tendon carried proximally over the proximal interphalangeal joint and then sutured to the extensor tendon where its lies on the proximal phalanx. It is seen that a sling is formed by the split proximal end of the paralysed tendon of the *flexor profundus digitorum*. This sling encircles the proximal phalanx; its function is to stabilize the proximal phalanx so that the middle phalanx may be extended upon it; the middle phalanx in its turn now forms a stable base for the extension of the distal phalanx.

The distal free end of the transversely divided tendon of the *flexor digitorum profundus* has been carried in its subdivisions around the middle phalanx and across the proximal interphalangeal joint; therefore, when the *flexor digitorum sublimis* acts, flexing the middle phalanx at the proximal interphalangeal joint, the transplanted tendon is necessarily dragged upon and the distal phalanx flexed.

There has been no interference with the long extensor which acts more effectively because muscle balance has been restored.

This little operation appears to the writer to embody much that is called for in orthopædic treatment. There has been no destruction of active functioning tissue; maximum use has been made of paralysed tissue in stabilizing one joint and in producing movement in another; the only functioning tissue at hand has been made use of to produce effective movement in three joints where previously there was absence of effective movement.

The great difficulty met with in attempting to improve the functional value of the two inner fingers in old cases of ulnar paralysis is not the carrying out of the operation just described; it lies in overcoming the contracture that has occurred in the interphalangeal joints and their neighbourhood. Hence the indication appears to be to perform this operation at an early period. Certainly if that had been done during the war, the functional value of the hand would have been increased in all cases.

The writer, however, is not prepared at present to advocate it as a routine early procedure. It appears to him that the results obtained today by conservative methods do not represent the best to be expected. He cannot help thinking that there is a great deal of harmful meddlesome interference in the handling of these delicate little structures, the paralysed muscles, and in attempts at so-called reeducation of them.

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## ENURESIS.[1]

By Edgar H. M. Stephen, M.B., Ch.M. (Sydney),
*Honorary Physician, Royal Alexandra Hospital for Children, Sydney; Lecturer in Medical Diseases of Children, University of Sydney.*

The term enuresis cannot fairly be ascribed to children until they have reached their third year. The condition rarely persists beyond the age of ten years and usually is a thing of the past long before this. Diurnal incontinence is unusual and almost always yields readily to training, sometimes assisted by drug treatment. We must all admire the infant of twelve months who, carefully trained by its parents, succeeds in keeping itself and its bed dry all night. On the other hand no one is likely to bear any rancour against the child who is less successful until the age of three years is attained.

It is essential that the patient with enuresis should have a careful examination made of the natal cleft. In the female a urethral caruncle (very rare) or nævus may exist. Various adhesions involving the labia and clitoris may be seen. Frequent and painful micturition, leucorrhœa, local irritation or pyelitis may be associated with enuresis in these cases. Once the condition is corrected, the enuresis is soon cured. Next inquire for possible thread worms and look to see whether any are present. Suggest an examination of the vulva and anus while the child sleeps. An enema of salt and water may be required to reveal an abnormal amount of mucus and threadworms. Even if the worms have not invaded the female genitals, the irritation and restless nights they cause are often responsible for an enuresis which disappears on the reluctant departure of the *oxyuris vermicularis*. The male child may have an unusually long prepuce and though freely movable, enuresis may be present until circumcision is performed. A tight prepuce, retained smegma and careless toilet are frequently the cause of enuresis and circumcision proves a necessary step in promoting a cure. Inquire into the diet. An ill-balanced diet may be responsible for the condition. A reduction in carbohydrate intake, especially of sugar, potato and raw fruit, may prove most beneficial. Perhaps the diminished acidity of the urine that follows this step may be the chief benefactor. In regard to examination of the urine, *diabetes mellitus* or renal glycosuria may be detected from the development of nocturnal enuresis. An over acid urine or that from pyelitis may be the cause. A stone in the urinary tract demonstrates its presence by the more striking array of pain, hæmaturia and pyuria. In the vast majority of cases examination of the urine is conscientiously performed without the acquisition of any gross information such as the above. Enuresis is more commonly a functional disturbance. The nervous child demonstrates

[1] Read at a meeting of the Pædiatric Section of the New South Wales Branch of the British Medical Association on August 24, 1928.

his type by this troublesome symptom. In my practice it has been the child with nasal obstruction, the snoring, mouth-breathing, ear-discharging, stale-breathed child who required removal of tonsils and adenoids that was the commonest sufferer from enuresis. An improvement in nerves and a dry bed were amongst the gratifying results of the surgeon's attention.

After exclusion of the above satisfactory types there are still some who require prolonged care and implicit instructions such as these: (i) Train the child to hold water as long as possible by day; (ii) do not give tea or coffee; (iii) give nothing to drink after 4 p.m.; (iv) arrange that the child is "held out" when the parents retire; (v) a flannel sleeping suit and a blanket bed conserve warmth and assist success; (vi) use cold bathing unless specially contraindicated. It is a stimulating process that can be recommended for most.

Now for methods with which I am entirely unfamiliar. Stern counsels or even castigation: these customs seem to me as unpleasing and inadvisable as the making of puns. The promise of rewards: it is said that in America some boys will keep their bed dry for five cents; they might raise their prices and this method become possible for the wealthy only. It seems to me a means unlikely to be effectual and suggestive of royal commissions. Drug treatment: the use of belladonna is often a success. Start with 0·6 mil (ten minims) of the tincture for the three-year-old at bed time. Supplement with half doses twice or thrice daily if necessary. The amount of the drug that just causes some dryness of the mouth should be the most required for an effective dose. As regards other drugs, an occasional success is scored by bromides given at bedtime, when a restless, dreaming child is induced to slumber peacefully. Strychnine may improve general tone and add to the prospect of success. I have had no experience of *tinctura lycopodii* given in similar doses to belladonna, *extractum rhus aromatici fluidi* or liquid extract of ergot given three times a day in 1·2 mil (twenty minim) doses. I would like to add that enuresis is sometimes a substitution phenomenon in epilepsy. This point Dr. A. W. Campbell told me of. Another point of some professional elegance is that enuresis may be due to vagotonia, that condition in which the autonomic system is in preponderance and the sympathetic in abeyance. In this condition adults are liable to asthma and eosinophilia and children to enuresis and *laryngismus stridulus*. Adrenalin administered by hypodermic injection at bedtime may relieve as in asthma. Beaumont and Dods's "Advances in Medicine" provided this information.

### Conclusions.

1. The presence of nasal obstruction with its consequent restlessness is a very frequent cause of enuresis. The necessary operation is followed by great improvement.

2. The presence of threadworms ranks next in importance as a cause.

3. The rectification of phimosis and structural defects of the external genitals must not be neglected.

4. The training of the child, cold bathing and warm bed equipment should be advised in detail.

5. If drug treatment is required to assist recovery, tincture of belladonna or an atropine solution are worthy of administration, the dose to be adjusted to the requirements of the individual patient.

## APPENDICITIS IN CHILDREN.[1]

By P. L. Hipsley, M.D. (Sydney), F.C.S.A.,
*Honorary Surgeon, Children's Hospital, Sydney; Honorary Surgeon, Royal Hospital for Women, Paddington.*

Since I am allowed only a short time to deal with the large subject of appendicitis in children, I shall have to confine myself to one or two aspects of the disease only. Someone has truly remarked that "the first part of treatment is diagnosis and the second diagnosis and the third diagnosis."

Once a correct diagnosis of appendicitis has been made, the treatment is comparatively simple.

There were 237 children, between the ages of twelve months and fourteen years, operated on for appendicitis at the Royal Alexandra Hospital for Children during the year 1927. Six of these children died.

The ages of the children were: Between twelve months and six years, 50; between seven and ten years, 116; between eleven and fourteen years, 71. The six deaths occurred in children whose ages ranged from four to seven years.

Now, although no doubt all these children had symptoms of appendicitis, I feel sure that not all the appendices removed manifested pathological lesions. We must therefore first consider what is generally understood by the term appendicitis. No doubt what is commonly called appendicular colic, is due to some abnormality of the appendix apart from an inflammatory lesion. The appendix may be dilated at the tip and constricted at the base or it may be kinked or adherent and on removal the pathologist would not be able to find any actual pathological lesion. Such appendices would give rise to colicky pain, if in the act of getting rid of fæces or masses of thread worms. Such appendices are certainly abnormal and, if causing pain, should be removed. No doubt many of the children operated on had abnormal appendices of this kind, but strictly speaking they would not be considered to be suffering from appendicitis. It is surprising how often one finds numerous thread worms in an appendix after removal and such appendices may show no pathological lesion to the naked eye, yet the pain necessitating the operation was undoubtedly caused by the organ trying to rid itself of the thread worms.

[1] Read at a meeting of the Pædiatric Section of the New South Wales Branch of the British Medical Association on August 24, 1928.

Then there is the vexed question of the nature of so-called chronic appendicitis. Frequent attacks of appendicular colic may lead to a thickening of the wall of the organ, but this can hardly be described as chronic inflammation.

Probably most cases of appendicular colic, from whatever cause, will, unless the patient is operated on, eventually become true cases of appendicitis. Therefore, in children at any rate, all such patients should be submitted to operation.

### Diagnosis.

Before considering the differential diagnosis of appendicitis in children I would like to make a few general remarks about the diagnosis of this complaint. I think that the error of diagnosing appendicitis when it does not exist, is due to the fact that the complaint is so common and that the diagnosis is so often made in a hurry before the evidence is carefully weighed. We then become insensibly biased and fail to see signs and symptoms which should be obvious. The blame for a late diagnosis often rests with the parents. Children suffering from stomach ache and vomiting are often not considered to be suffering from anything out of the ordinary and medical advice is frequently not sought early enough. In my opinion recurring attacks of abdominal pain in children are more often due to appendicitis than to any other cause.

In the diagnosis of disease in children we have to rely mainly on objective signs rather than on symptoms. In cases of doubt a rectal examination should always be made, as the appendix may project downwards towards the pelvis; in such cases only very firm pressure above Poupart's ligament will elicit tenderness.

A leucocyte count is of value in acute cases which are generally characterized by a leucocytosis. Robert Hutchison speaks of the right iliac fossa as a region of the body in which mare's nests are particularly common. In other words it is a region where signs of disease which have no real existence, are so commonly found. This, however, applies to adults rather than to children. Fortunately in children we can eliminate from the diagnosis many conditions which are common in adults, such as visceroptosis, duodenal ulcer, acute pancreatitis, cholecystitis, acute salpingitis and ectopic gestation. But after disregarding all these, we still have a list of pitfalls which give rise to frequent mistakes in children.

#### *Affections of the Urinary System.*

I have seen several children whose appendices have been removed when the real trouble has been hydronphrosis or pyonephrosis. Renal or ureteral calculus is often mistaken for appendicitis. Pyelitis is also commonly diagnosed as appendicitis. This complaint is common in young girls, rarer in boys. The temperature is irregular and often associated with rigors in older children and with convulsions in young children. A microscopical examination of the urine will reveal the trouble and should never be omitted in suspected appendicitis in young girls. On the other hand I have seen a patient with appendicitis treated for many months as though suffering from cystitis, owing to the fact that the inflamed appendix was adherent to the bladder. I can recall another case in which a patient with a retrocaecal and gangrenous appendix was treated for months as though he was suffering from renal colic.

#### *Appendicitis Resembling Acute Pneumonia or Pleurisy.*

The mistake of diagnosing acute pneumonia as appendicitis is not uncommon. This is owing to the fact that the abdominal muscles are on guard in both conditions and abdominal pain is often present in pneumonia. The flushed face, rapid respirations and signs in the chest should make the diagnosis clear. In case of doubt, in the early stages a leucocyte count might help, as it is generally higher in pneumonia.

#### *Pneumococcal Peritonitis.*

There are several varieties of pneumococcal peritonitis, varying from the acute fulminating to the chronic, and each variety may be mistaken for appendicitis. The condition is almost confined to girls, generally of about five or six years of age. The abdominal pain is more generalized and there is not the localized tenderness and rigidity in the right iliac fossa in the early stages. Signs of a general blood infection soon appear and the condition becomes one of pneumococcal septicæmia with signs of general peritonitis. The children are often cyanosed quite early.

#### *Congenital Malformations of the Mesentery.*

Congenital malformation of the mesentery is sometimes the cause of obscure abdominal pains, coming on periodically and associated with vomiting; it is often diagnosed as appendicitis. The whole of the small bowel in these patients is often found on one side of the abdomen and this will give a fullness on that side with a corresponding feeling of emptiness on the other. A bismuth meal followed by an X ray examination five or six hours later and a rectal injection of bismuth given at the same time that the X ray picture is taken, will reveal the trouble.

#### *Henoch's Purpura.*

Henoch's purpura is associated with abdominal pain and vomiting and so may be mistaken for appendicitis. There are generally, however, other signs present, such as pain and swelling of joints and subcutaneous or submucous hæmorrhages. When these are absent, the diagnosis is not always clear at first. Sometimes a mass due to hæmorrhage in the intestinal wall can be felt and this may further obscure the diagnosis.

#### *Cyclic Vomiting.*

Cyclic vomiting which is so common in children, is often diagnosed as appendicitis. In this condition abdominal pain is not so pronounced nor do we get the localized tenderness and rigidity that are so characteristic of appendicitis. The abdominal wall

in cases of cyclic vomiting is more often flat or retracted and the incessant vomiting is out of all proportion to the abdominal pain. The presence of acetone in the urine is important, but too much stress should not be placed on this fact, as acetone is so often found in the urine of children vomiting from other causes or indeed when there has been no vomiting at all. Although there may be fever in cases of cyclic vomiting, the temperature is often normal or only slightly raised.

*Meckel's Diverticulum.*

The persistent remnant of the omphalo-mesenteric duct known as Meckel's diverticulum may cause trouble, giving rise to acute abdominal pain and vomiting in one of two ways. First when it is free in the abdomen, an intussusception may start in it and, second, when it is adherent to the umbilical region, it may cause obstruction and strangulation of the bowel. I have seen examples of both these conditions. The intussusception occurred in a girl of six years of age and the strangulation of the bowel in a boy aged eight years.

One must bear in mind in these cases the difference between the symptoms in inflammatory lesions of the bowel and those in obstructions and strangulations. E. R. Flint[1] summarizes the difference in the symptoms of these conditions as follows. He states:

> In inflammatory lesions we get pain, localized tenderness and localized rigidity, vomiting, quickened pulse and raised temperature. In obstructions and strangulations we get pain, very slight if any tenderness, no rigidity, persistent vomiting, rather quickened pulse, temperature around normal and absolute constipation.

In other words, the absence of rigidity and of fever and the presence of persistent vomiting in cases of strangulation should make the diagnosis clear.

*Hysteria.*

Friedjung, quoted by Stern,[2] in 1904 described a typical form of hysteria in children between the ages of three and ten years. It is characterized by sudden attacks of pain in the umbilical region. The pain lasts for fifteen or thirty minutes and is not usually associated with vomiting. The bowel movements are generally normal and the patients have a hyperæsthesia of the skin of the abdomen and over the cervical and thoracic vertebræ.

> A typical sufferer from this complaint was admitted to hospital under my care a few months ago with a diagnosis of acute appendicitis for immediate operation. The patient was a girl aged ten years. She had a very small umbilical hernia which was obviously not causing any trouble and I could find no signs of appendicitis except the pain.

*Intussusception.*

It is only when intussusception occurs in older children that mistakes in diagnosis are likely to occur. I have seen several chronic intussusceptions in boys of six or seven years of age and owing to the attacks of abdominal pain, associated with vomiting, appendicitis had been diagnosed. These chronic intussusceptions frequently do not cause complete obstruction as occurs in acute intussusception. The absence of fever and of rigidity and the presence of a movable swelling in the right iliac region should be sufficient warning of the possibility of this condition being present.

*Typhoid Fever.*

Owing to abdominal pain, high temperature, localized tenderness and rigidity over the right iliac fossa, typhoid fever is occasionally mistaken for appendicitis.

*Spinal Caries.*

Long continued or frequently recurrent attacks of abdominal pain are sometimes caused by caries of the spine in the lower dorsal region.

*Catarrhal Jaundice.*

Owing to the abdominal pain, fever and vomiting in the early stages of catarrhal jaundice and before the jaundice is very noticeable, it is not uncommon for it to be diagnosed as appendicitis.

*Abdominal Colic from Overeating.*

Lastly, abdominal colic from overeating, both in children and adults, has, I fear, been responsible for the removal of many innocent appendices. In suspected cases a dose of castor oil and an enema will clear up the diagnosis. At the same time the indiscriminate giving of castor oil to children on the slightest pretext is to be deprecated, as it may cause extension of the inflammation in early cases of appendicitis. J. C. Motley[3] found that out of nineteen patients admitted to hospital when the appendix had perforated, no less than sixteen gave a history of free purgation.

**References.**


[1] E. R. Flint: "An Address on the Acute Abdomen," *The British Medical Journal*, February 11, 1928, page 209.

[2] A. Stern: "Results from Finkelsteins' Casein Milk in Twelve Children with Chronic Gastro-Enteritis and Marasmus," *Journal of the Medical Society of New Jersey*, 1910, Volume VII, page 408.

[3] J. C. Motley: "Appendicitis in Children," *The Journal of the American Medical Association*, November 4, 1916, page 1364.


# Reviews.

## ERRORS IN REFRACTION AND THEIR CORRECTION.

In "The Prescribing of Spectacles," by A. S. Percival, a book of 239 pages, we are given the fruits of a long personal experience in correction of refraction and muscle balance of the eye.[1] The work commences with a short history of lenses and the theories of accommodation.

The reasons for change of refraction in old age and the changes in accommodation are carefully dealt with. In actual testing of refraction the theoretical tests are shortly

[1] "The Prescribing of Spectacles," by Archibald Stanley Percival, M.A., M.B., B.C. (Cantab.); 1928. Bristol: John Wright and Sons, Limited. Crown 8vo., pp. 239. Price: 15s. net.

disposed of. The important matter of subjective examination is given in full. Retinoscopy is completely dealt with from a practical point of view and the use of cylinders fully described. The great accuracy and value of this method is pointed out. The function of the ophthalmometer and tables for its use are given. The common incorrect statements in text books on anisometropia have been exposed in the light of the author's experience. The space devoted to periscopic lenses is more than usual and very interesting, but expense is a factor which precludes their use in routine work. The special rectangular wafer which is recommended for use in bifocals does not seem to have come into favour with the manufacturers, although at least one ophthalmic surgeon in London claims increased personal comfort from them. In treatment of high myopia there is a timely warning against bifocal lenses. Prism treatment of heterophoria is particularly well discussed and it is clearly indicated how and when prisms should be ordered. The author quotes from special cases and tells exactly how he treated the patients. This section alone would recommend the book to many ophthalmologists. There are short notes on symptoms and their cause and also on frame fitting. The optical section would no doubt appeal to those with a mathematical turn of mind. It is a book well worth reading and possessing.

## DIETETICS.

IN the introduction to a book called "Handbook on Diet," by Dr. E. E. Marcovici, of New York, is found the following statement: "This book, it is hoped, will prove to be simple enough in its terminology and yet of sufficient accuracy to warrant the physician in recommending its perusal to his patients."[1] The book, however, is somewhat too technical for patients' use. It is not wise to hand to patients a book containing dietetic rules and diet lists for a variety of diseases. A shorter and more general work on normal diet and principles of dietetics would seem more valuable and the diet in an individual disease, such as *diabetes mellitus*, might better be dealt with in a book dealing with such complaint from other aspects as well. However this may be, this book is on the whole accurate and could certainly be recommended to medical practitioners and students; they will be certain to pick up many points of value, whatever their knowledge of dietetics.

The section on *diabetes mellitus* exhibits a certain lack of coherence, there being no proper correlation between modern knowledge and its practical application. This is probably because the author has not entirely assimilated the facts concerning protein and fat metabolism which have revolutionized the treatment of *diabetes mellitus*. These facts have rendered fallacious the use of large quantities of protein as advocated by Van Noorden, to whom the author was an assistant in Vienna, and incidentally have shown that "fast days" which are also advocated by the author, are unnecessary in all cases and actually harmful in some.

In Chapter II, called "Food; Its Utilization in the Body," there is a short table showing the correlation between the number of calories in the diet on the one hand and weight and activity on the other. The number of calories recommended is as follows:

30 to 35 calories to 1 kilogram body weight daily at full rest.
35 to 40 calories to 1 kilogram body weight daily at slight work.
40 to 45 calories to 1 kilogram body weight daily at average work.
50 to 60 calories to 1 kilogram body weight daily at hard work.

In the actual diets listed in later chapters, even for *diabetes mellitus*, there is little practical application of this principle.

Sippy's and Lenartz's diets are not mentioned in the description of the diet for gastric ulcer. In dealing with gout and later with salt retention in kidney and heart diseases, the author urges that the tolerance for purin bodies and for salt should be measured just as carbohydrate tolerance is often measured.

"Thirst days" are described in Chapter VII and recommended for inflammatory rheumatism, general dropsy, tuberculous dropsy, cirrhosis of the liver and psoriasis. These "thirst days" may take the form of restriction of fluids to about four hundred cubic centimetres with liberal allowances of cereals and proteins. Oertel's ideas on "thirst days" are indicated. He advocates the method in the treatment of disturbance of the circulatory system. He considers thirty grammes of water per kilogram of body weight as the daily requirement under normal conditions and gives twenty, fifteen or even ten grammes per kilogram according to the condition.

To the list of omissions already mentioned and that of liver diet in pernicious anæmia, there are few to add. Vitamins are rather inadequately dealt with, however. The book contains long lists of recipes, tables of food analyses and two chapters on mineral waters and bath resorts in the United States of America and Europe.

Altogether its three hundred pages contain a great deal of information and the book is singularly free of inaccuracies. It is a book worth purchasing, for it would be used often on account of its distinctly practical attitude to the subject. If it is a fact that books are the tools of the mind, this is one to put in the workshop.

## A BACTERIOLOGICAL MANUAL.

THE second edition of Mackie and McCartney's "Practical Bacteriology" is a very great improvement on the previous edition. One hundred pages of new matter have been added and the whole book has been practically rewritten.[1] The early chapters give a more complete account of culture methods and the use of the microscope and a very clear discussion of immunity. In addition to the pathogenic organisms of man, those of the ordinary domestic animals are described with their relation to allied diseases in man. In the discussion of diphtheria and scarlet fever the authors have given a description of the Schick and Dick reactions and of toxin-antitoxin immunization. Special attention is paid to laboratory methods of diagnosis and this practical book is thereby made a more complete guide for students and practitioners A few line drawings have been introduced to facilitate description of apparatus and methods. These could with advantage be increased in number.

The book is sound and essentially practical. For the laboratory worker and for the practitioner who is required to carry out occasional bacteriological tests, it is an essential addition to the library.

## PRESENT DAY KNOWLEDGE ON TOBACCO.

IN "Tobacco and Physical Efficiency" Professor P. Schrumpf-Pierron provides the key to an enormous literature bearing on the pharmacological, physical and psychological effects of tobacco.[2] The book consists of two parts of almost equal length. The first is a succinct summary and the second an annotated bibliography. The summary is indeed succinct to the point of terseness, but serves as a guide to those publications which a worker on the subject would find useful. There has been no attempt at critical estimation of the value of the contributions indexed, but as a bibliography the book should fulfil a want.

[1] "Handbook on Diet," by Eugene E. Marcovici, M.D.; 1928. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 330. Price: $3.50 net.

[1] "An Introduction to Practical Bacteriology: A Guide to Bacteriological Laboratory Work," by T. J. Mackie, M.D., D.P.H., and J. E. McCartney, M.D., D.Sc.; Second Edition; 1928. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 404. Price: 10s. 6d. net.

[2] "Tobacco and Physical Efficiency: A Digest of Clinical Data," by Pierre Schrumpf-Pierron, M.D.; 1927. New York: Paul B. Hoeber, Incorporated. Demy 8vo., pp. 147. Price: $1.85 net.

The Medical Journal of Australia

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## The Annual Meeting of Delegates.

The New South Wales Branch of the British Medical Association has for many years adopted an expedient whereby the views and opinions of members practising in the several parts of the State can be ventilated, expounded and discussed. The annual meeting of the delegates of the local medical associations with the Council of the New South Wales Branch was held on October 5, 1928, and as in former years this meeting proved to be an admirable means of overcoming a difficulty. The local medical associations, like the analogous groups or societies in other States, are bodies formed outside the British Medical Association. Their membership, however, is limited to members of the Branch and they stand in close relationship with the Branch. Their position is somewhat anomalous; while they have no constitutional existence within the British Medical Association and a member of the latter body resident in the area of a local medical association cannot be compelled to join it, they carry out many of the functions performed by the Divisions of the British Medical Association. It is obvious that the medical practitioners residing in a limited area are more competent to define the peculiarities of medical practice in that district and to determine the most expedient measures to meet local difficulties than any centrally situated body. It is evident from the annual discussions that the problems of hospital practice, of friendly society lodge practice and of medical treatment given in accordance with the provisions of the *Workers' Compensation Act* vary not inconsiderably in country, suburban and urban districts. The man on the spot with first hand experience is necessarily a better judge of these things than the man whose personal knowledge of rural affairs dates back several years. The local medical associations possess sufficient machinery to enable their members to handle local matters, but they are not uniformly active bodies and at best they are not powerful enough to act collectively with outside bodies. It is to the Branch of the British Medical Association and to the Federal Committee that the members have to look for the formation of policies and for the moulding of medico-political action. In the circumstances the local medical associations are fortunate in being affiliated with the New South Wales Branch and in having an annual opportunity of free discussion with the Branch Council. The delegates, it is true, vote without any actual power to instruct the Council to give effect to their wishes. As the local medical associations have no constitutional existence within the Association, the annual meeting of delegates serves the purpose of informing the Council concerning the views held in the several parts of the State and thus of guiding the Council.

It is seemingly not clear to every one why greater powers and more latitude cannot be given to the local medical associations. Members appear to forget that the administrative unit of the British Medical Association is the Division. In the United Kingdom each Division enjoys a large degree of autonomy. For certain defined purposes of representation, of monetary distribution and of collective action, a group of contiguous Divisions is formed into a Branch. Although the Branch controls the purse to some extent and possesses some special powers, it has no real medico-political jurisdiction over the Divisions. The overseas Branches are Branch-Divisions and enjoy all the privileges and powers of the Divisions of the United Kingdom. They are the indivisible units. If the constitution of a Branch were amended to admit of the recognition of local medical associations as constituent parts of the Branch, these local bodies would usurp the powers and privileges of Divisions and the Branch Councils would be deprived of the greater part of their controlling influence. It would be necessary for the whole area of the Branch to be divided into Divisions and each Division would be granted relative autonomy. This would mean that the unique achievements of the Branches of the British Medical Association in Australia in regard to organization, membership and recognition would be endangered by the inefficiency of the secretarial

offices of the individual local bodies. Even the most enthusiastic advocate of local representation would hesitate to recommend the substitution of multiple secretarial offices for the powerful and well organized office in Sydney. It is obvious that unless the local associations were given a standing in the constitution of the British Medical Association, it would be both illogical and unconstitutional to give them representation on the Council of the Branch. However desirable it might be to grant the local medical associations the right to nominate or to elect representatives to the Branch Council, this cannot be done without a profound change in the constitution of the New South Wales Branch and perhaps of all the Branches in Australia. Permission could not be given by the British Medical Association for any constitutional change that is foreign to the constitution as set out in the Memorandum and Articles of Association. If the local medical associations sought recognition as Divisions, each and every one would have to make application and if there were a strong opposition to the proposed change, the Council of the British Medical Association would probably refuse permission.

The New South Wales Branch of the British Medical Association holds the proud position of one of the strongest and best organized Branches in the whole Association. Its members in the most remote corners enjoy all the privileges of a powerful institution that uses its authority judiciously and in a measured manner. It would be unwise and even disastrous to disturb the present position, more particularly because there are indications of further growth of the usefulness of the Branches in Australia to the members and to the community. The annual meeting of delegates is an admirable institution and suffices to temper the decisions of the New South Wales Branch Council with wisdom born of the messages delivered at this meeting.

## Current Comment.

### EXERCISE AND DIGESTION.

If the average man in the street were asked whether the taking of exercise immediately after a meal had any influence for good or evil on digestion, he would probably reply by quoting the old adage: "After dinner rest awhile; after supper walk a mile." If the average medical practitioner were asked the same question, he would be most likely to advise that exercise productive of no unpleasant symptoms was not likely to have any untoward effect. Both replies would meet the situation up to a certain point. But if an investigation of the literature be made as to what exact data there are dealing with the subject, it will not be so easy to set up definite standards.

It is therefore of interest to read a communication on the subject by J. M. H. Campbell, A. O. Mitchell and A. T. W. Powell.[1] They have observed the differences produced in the digestion of the same meal in a person at rest and after taking exercise. Their experiments were divided into two sets of observations. In one set experiments were carried out with a fractional test meal and in the other experiments were made with a one-hour test meal of porridge. In the first set two subjects took part in twenty tests after a light luncheon of meat, bread and potatoes. Sometimes they rested and sometimes they ran for a few miles and samples of gastric contents were withdrawn through a Ryle's tube each quarter of an hour after the meal. In these specimens the free and total acidity were estimated by titration with decinormal soda, dimethylphthalein and phenolphthalein being used as indicators. These experiments revealed changes after exercise in the acidity of the stomach contents and in the rate of secretion of gastric juice, but the rate of emptying was difficult to find, as it was impossible to withdraw the residue of the meal completely at the end of an hour. The percentage acidity, both total and free, at each half hour interval was less when the subject took exercise than when he was at rest. The slower rise in acidity when exercise was taken, was due to a diminished secretion of gastric juice. In regard to the rate of emptying the difference was insignificant; on an average starch disappeared after 1·9 hours with exercise and after 2·1 hours with rest. The quantity of the meal given was 25 grammes of bread, 25 grammes of lean beef and 50 grammes of potatoes. The results of eighteen experiments only are recorded and there is no indication as to which of these were carried out with a large meal or how large such a meal was. These "points of interest" are in keeping with the present day conception of hormonal control of digestion as enunciated by Starling. The chemical messengers call forth the secretion of gastric and other digestive juices in accordance with the requirements of any particular quantity of food ingested.

In the second set of experiments the meal given consisted of 50 grammes of oatmeal, 12 grammes of cane sugar and 800 cubic centimetres of water, mixed together and boiled down to half their bulk. The meal was given at midday after the stomach had been emptied. The stomach contents were withdrawn and measured one hour after the meal had been taken. The proportion of the original meal in the residue was found and the presence of bile, the free and total acidity and the chloride content were

[1] *Guy's Hospital Reports*, July, 1928.

determined. Ten pairs of experiments were made on seven subjects; each subject rested on one occasion and on another ran round the laboratory for part of the hour after the meal, the distance run (from one to four miles) depending on the degree of training. The results in these circumstances were much more striking than with the first set of experiments; when exercise was taken a higher percentage of carbohydrates and total solids was found, there was a lower percentage of acids and chloride and there was a larger gastric residue. When the subject had been at rest, the residue was generally between 20 and 190 cubic centimetres; when exercise had been taken, it was between 200 and 270 cubic centimetres. In the ten experiments with the subject at rest there were on an average 45 cubic centimetres of the original meal and 94 cubic centimetres of gastric and other juices. In the ten experiments in which the subjects took exercise, the amount of gastric juice remained the same (97 cubic centimetres), but it was mixed with three times the quantity of the original meal; the percentage of carbohydrate was thus higher and of acidity lower. This suggested to Campbell and his collaborators that exercise had delayed the rate of emptying, but had not diminished the secretion of gastric juice. They state, however, that presuming the secretion of acid to have been at the same rate, less would have been found in the stomach at rest, because more would have passed on through the pylorus with the more rapid rate of emptying. The amount of acid actually found was the same, since more of the gastric contents had left the stomach, more acid must have been secreted in the stomachs of the subjects at rest. It is doubtful whether the authors are justified in their assumption that the secretion of acid was at the same rate. Reference to their first series of experiments shows that the average percentage of free and of total acid was less during the first two hours in subjects who took exercise than in those who were at rest, except in one or two instances in which the figures at two and a half and at two hours were greater in the persons who took exercise. It would be difficult to prove that the contention was wrong that secretion was somewhat delayed in the earlier stages when the subject was exercising. In fact, later on in the article in discussing the second series, they show that the subjects naturally fell into two groups. Those who could run further and who were thought more fit, had a slower pulse rate after exercise, the difference being slight but constant. In all the men exercise "delayed the rate of emptying and the secretion of gastric juice," but the amount of exercise needed to produce this effect depended on their fitness. This is an apparent contradiction of the previous statement. At the same time it is unlikely that any delay in secretion would account entirely for the differences observed in the second series of experiments. Another factor to be kept in mind is the neutralizing effect of regurgitated duodenal contents. In the second series of experiments bile was present in some of the specimens taken from the subjects who had undergone exercise, showing clearly that regurgitation had taken place. This was not an important factor, because there was still a pronounced difference in the total chloride concentration.

Observations were also made on the effect of walking exercise and of hot baths. The effects of walking after the meal were "the reverse of those due to running"; the amount left in the stomach was less, though the secretion of gastric juice was little affected. When two persons walked and talked together, their stomachs emptied more rapidly than when they walked alone. It is concluded that exercise which does not produce discomfort of any sort, increases the rate of digestion, but that the opposite effect follows exercise which produces any discomfort. In view of the suggestion that cutaneous vasodilatation might be one factor influencing the stomach, the effect of hot baths was tried. When hot baths were used, gastric secretion was delayed, but there was no effect on the rate of emptying. When baths were very hot, gastric secretion was delayed as much as after running, though the rate of emptying was still not so much delayed.

Campbell and his collaborators thus arrive at the above mentioned conclusions which may be summarized as follows: The emptying time of the stomach is prolonged when exercise of a moderately severe or severe nature, such as running, is taken after a meal. When walking exercise, however, is taken, the emptying time is less than when the body is allowed to remain at rest. The greatest quantity of gastric juice was produced when the body was at rest. Light exercise did not delay the secretion of gastric juice and the percentage of hydrochloric acid in the gastric juice secreted was practically the same under all circumstances. Campbell and his collaborators, however, do not enter into any discussion as to the reason for any alteration in the part of the digestive process discussed by them as a result of exercise. They state that cutaneous vasodilatation with relative anæmia of the stomach probably provides the main mechanism by which the effects are produced. The suggestion that the higher centres have a definite effect demands consideration. Mental anxiety and worry have an influence on bodily function and it is easy to understand that a contented mind would make for the smooth running of the bodily machine. The products of muscular action would certainly operate on the vasomotor centre and possibly on the hormones concerned in digestion. It must also be pointed out that Campbell and his fellow workers have set out to discuss the influence of the taking of exercise on digestion, but have confined themselves merely to one phase of the process. Digestion includes the breaking down of foodstuffs into simpler components and their absorption in the circulation. If the full effect of exercise on digestion is to be measured prolonged observations in a calorimeter must be made of persons who take severe exercise after the ingestion of standard meals. An indirect method might be used by subjecting two groups of persons to prolonged observation under controlled conditions and observing their variations in weight.

# Abstracts from Current Medical Literature.

## SURGERY.

### Cleft Palate.

W. E. M. Wardill (*The British Journal of Surgery*, July, 1928) in a Hunterian lecture delivered before the Royal College of Surgeons of England on cleft palate, describes a new operative procedure. He states that in the usual operations performed for this condition the results have been eminently satisfactory as far as closure of the cleft is concerned, but that when restoration of normal speech is considered, these operations have been a lamentable failure. If the operation of gastro-enterostomy had to rely for its popularity on such slender results as are obtained in cleft palate, the patients who would submit to such a procedure, would be very few and far between. The sufferer from cleft palate after surgical treatment is unlikely to become a normal speaker. He may have nasal speech with an intact hard and soft palate. A skilled dental surgeon by means of an obturator can do as much as this. In considering the anatomy of speech, the importance of the muscular tissue on the posterior wall of the pharynx, constituting Passavant's ridge, is emphasized. The superior constrictor muscle contracts and elevates the ridge of Passavant, thereby narrowing the naso-pharynx antero-posteriorly and also from side to side. It remains in this state of contraction throughout speech and only when speech ceases, does it relax. This forms a buffer against which the highly mobile soft palate can flap. Normal speech depends upon the competence of this valve between the soft palate and Passavant's ridge. It is upon this factor and not upon a perfect anatomical closure of the cleft, that the restoration of normal speech depends. Cleft palate speech depends on two factors: the incompetent naso-pharyngeal valve and incorrect speech habits designed to disguise the incompetence. The letters "k" and "s" are particularly instanced and the training of a cleft palate speaker is likened to a normal speaker learning a new language. An attempt to form a naso-pharyngeal valve must be the main aim of an operation to restore speech; if this is not done and the cleft merely closed, the result is analogous to the fitting of an obturator. In considering the anatomy, it is certain that the two halves of a cleft soft palate are not together equal to a whole normal soft palate. Defects in the skull bones are more obvious than those in the soft tissues. In many cases of unilateral cleft there is gross asymmetry of the calvarium. The parietal eminence on the side of the cleft in many specimens is found to be considerably displaced further backwards than its opposite fellow. There is also a definite increase in the transverse diameter of the naso-pharynx. The surgical problem thus resolves itself into the formation of a new naso-pharyngeal valve. Working on the principle that as the soft palate cannot be brought to the posterior pharyngeal wall, the posterior pharyngeal wall must be brought to the soft palate, the author has devised the following two-stage operation. At the first stage the mucous membrane over the ridge of Passavant is incised transversely, care being taken not to injure the bucco-pharyngeal fascia. This incision is then sewn up in a vertical direction. This narrows the naso-pharynx and at the end of the operation the halves of the soft palate are found to be much closer together and the halves of the uvula may overlap. The second stage is done after an interval of about three or four weeks and consists of either a Langenbeck-Fergusson or Gillies-Fry operation on the soft palate. Nine cases are reported with one death. The dangers of the operation are stated to be mediastinitis, asphyxiation and deafness. Mediastinitis is due to perforation of the bucco-pharyngeal fascia, asphyxiation may occur after operation and is guarded against by a suture being left in the tongue. Deafness was complained of by two patients after the second stage, in one it was transitory, in the other slight deafness persisted.

### Spinal Cord Surgery.

Ernest Sachs (*Surgery, Gynecology and Obstetrics*, September, 1928) in an address on spinal cord surgery gives the following indications for operation: Fractures and dislocations; inflammatory processes, meningitis, abscess or solitary tubercles; compression of the cord due to deformities; spasticities due to inflammatory diseases, cerebral birth palsies or old injuries; spinal tumours; relief of intractable pain and *spina bifida*. In simple fracture of the spine no operation is indicated, if no cord symptoms are present; when these are present, the question of operation should be determined by the Queckenstedt phenomenon whether any spinal block is present. If block be present, early exploration should be undertaken. If there is no block, it is probably useless in most instances to operate. In meningitis due to pyogenic organisms the lumbar region should be drained at the level of the third or fourth spinal process. With this method there have been six recoveries in twenty-two cases. Most instances of cord compression due to spinal deformity are the result of Pott's disease and ample opportunity should be given to the patient to recover with orthopædic measures. After a fair trial of about six months, if there is no improvement, operation should be performed, care being taken not to open the *dura*, if the process is extradural. In spastic paraplegia Foerster's operation still remains a most helpful procedure in selected cases. The tumours, being fibromata and endotheliomata, are usually benign. The chief difficulty in these conditions is the diagnosis and localization; this is aided by the Queckenstedt phenomenon and "Lipiodol" injections, the latter being used only when other methods fail. The treatment of extramedullary tumours is less satisfactory. Blood vessel tumours may be encountered which present atypical symptoms and extremely difficult operative problems. For intractable pain due to metastases of malignant disease or to tabes, the Spiller-Frazier operation of chordotomy may be a great comfort, though it does not always bring relief and in some instances, if carried out too deeply, may cause some paralysis. The various types of *spina bifida* also call for operative interference. When unruptured and when there is some evidence of some nerve fibres passing into the lower extremities or some rectal control, the patient should be operated on at an early age. A considerable percentage developes hydrocephalus. The aim is to reduce the hernial contents, close the *dura* and repair the defect with a fascial flap. Some instructions are given in the technique of operations on the spinal cord.

### Catheterization of the Ejaculatory Ducts.

Georges Luys (*Bulletins et Mémoires de la Société des Chirurgiens de Paris*. December 2, 1927) describes in detail the technique and indications for catheterization of the ejaculatory ducts. Metallic stylets are passed through a urethroscope. The indications given are: stricture of the ejaculatory ducts, vesicular retention when massage of the vesicles gives no result, painful ejaculation, hæmospermia, powerless ejaculation, congenital strictures of the ejaculatory ducts, relapsing epididymitis and especially azoospermia.

### "Insulin"-Glucose Treatment of Traumatic Shock.

E. C. Padgett and T. R. Orr (*Surgery, Gynecology and Obstetrics*, June, 1928) discuss and make an experimental report on the "Insulin"-glucose treatment of traumatic shock. There are indications that the chief factor in the production of traumatic shock is a toxin developed in damaged tissue. The toxic agent is unknown, but histamine is an example of a protein cleavage product which produces experimentally the classic picture of shock. It is probable that histamine does not represent the only example of a protein autolytic toxin which will produce shock-like symptoms. In primary shock the symptoms appear to be the result of a powerful stimulus of nervous tissue which results in a reflex relaxation of the vascular tree. Secondary shock is initiated and due principally to a toxic factor which increases the permeability of the capillary walls, resulting in a decreased blood volume and a falling blood pressure. Because of the rapid loss of blood volume through the capillaries Cannon suggests the term "exæmia" as being descriptive of the essential state of the circulatory apparatus in shock. In the interpretation of shock treatment it must

be remembered that patients with mild shock often recover either with or without the use of the common intravenous solutions. It must, too, be definitely determined that the condition dealt with is really shock. Hypertonic solutions of crystalloids have not been used with complete success in the past in the treatment of well developed shock. Recently evidence has been accumulating that intravenous therapy with hypertonic solutions of crystalloids, unless introduced with the greatest care, may be actually harmful. From the experimental standpoint the "Insulin"-glucose treatment of shock, recently introduced by Fisher, seems to be no more beneficial than treatment with a hypertonic solution of glucose or sodium chloride. Further studies should be made to discover, if possible, whether a patient in the true shock state can utilize glucose with "Insulin," as do normal individuals or patients suffering with other diseases.

### Radiology and Surgery in Breast Cancer.

Douglas Webster (*The Lancet*, July 14, 1928) discusses the respective positions of radiology and surgery in cancer of the breast. It was once true, but is true no longer, that the only chance for the patient with cancer of the breast is early operation. By radiology alone or as the main treatment results are being obtained in cancer of the breast similar to those in cancer of the cervix; a certain percentage of patients is cured and the results in the operable group are rather better than those from surgery alone. Combined measures of radiation and operation offer a fruitful field for further advances. Radiation for artificial menopause is little used, but is possibly a valuable auxiliary treatment. Radiological methods stimulate and also encourage the natural attempt at cure. Experimentally radiated areas have been shown to have an immunity to tumour implantation. This is an additional argument in favour of pre-operative and post-operative radiation. Operative removal is sometimes followed by the rapid growth of previously latent secondary deposits, possibly by loss of a "growth restraint" of the primary growth (Ewing). Experimental and clinical experience has shown that the best method of stimulating a growth appears to be to cut into it. Clinically this has been seen in sarcomata and the inflammatory type of breast carcinoma. In the hope of avoiding a considerable percentage of recurrences after incomplete operation, there is a very strong case for invariable pre-operative and post-operative radiation of the operation area and of the adjacent lymphatic drainage areas. No operations should take place on live unsterilized tumours. The ideal method is for all new patients with malignant disease to be seen by the surgeon and radiologist in consultation. As a rule the purely surgical view prevails and radiotherapy is considered as merely an adjuvant to surgery, to be employed only when surgery has failed. Radiotherapy has become a new specialty and the radiotherapist should be given a status of real equality.

### Symptoms Produced by Distension of the Gall Bladder.

V. L. Schrager (*Surgery, Gynecology and Obstetrics*, July, 1928) writes on his observations and experiments on the symptoms produced by distension of the gall bladder and biliary passages. It is observed that in certain instances biliary colic is associated with respiratory embarrassment at the height of the attack. This occurs with a distended gall bladder or with stone impacted in the cystic duct. This symptom has been of much value in the differential diagnosis of diseases of the gall bladder and biliary ducts, because it is either absent or insignificantly present in any other abdominal complex. Experiments have demonstrated that distension of the gall bladder or biliary ducts causes inhibition of respiration, chiefly inspiratory in type. There are also produced nausea, vomiting and distress, in proportion to the degree of distension. Distension of the biliary passages causes more striking symptoms than distension of the gall bladder alone. These symptoms are abolished by section of the vagi and some respiratory inhibition is likewise abolished by section of the right splanchnic. Instillation of "Procaine" or cocaine into the bile ducts temporarily abolishes or ameliorates the symptoms caused by distension. This is also true of a subcutaneous injection of cocaine. Blocking of the right splanchnic nerve with "Procaine" or alcohol under direct vision produces results equivalent to right splanchnic section. Distension of the ducts causes changes in the blood pressure and heart rate, but these changes are not uniform and are probably dependent upon the functional condition of the cardio-vascular system at the time the distension occurs. Intermittent distension of the biliary passages over a period of one week caused decided dilatation of them. It is further observed that during the respiratory inhibition caused by distension, the right side of the diaphragm was contracted apparently to serve as a splint.

### Surgery of the Knee Joint.

Arthur Krida (*The American Journal of Surgery*, July, 1928) gives his conclusions drawn from a review of fifty-five operations and discusses the surgery of the knee joint. In many chronic disabled knee joints the cause of the persistence of symptoms is contained within the joint itself. The end products of gross traumatism or of infection are little, if at all, influenced by extrinsic measures, such as removal of foci of infection. The retention of such end products within the joint, by precipitating a vicious cycle of traumatism and joint irritation, leads to gradual deterioration of the joint. The removal of hypertrophic and obstructing tissue at a stage before joint deterioration is too advanced, checks this process and results in the restitution of joint function. In suppurative arthritis operated upon at a favourable time, ankylosis may be avoided by complete excision of the infected synovia. Joint function cannot be preserved in the presence of gross deterioration of articulating surfaces or gross invasion by granulating tissue. Such joints are fit only for arthrodesis. Such operations cannot be satisfactorily conducted through small incisions and adequate exploratory incisions are not accompanied by any additional hazard.

### The Treatment of Burns.

F. Christopher (*The American Journal of Surgery*, July, 1928) presents an account of the modern methods of treatment of burns. Patients with extensive burns, that is those involving 10% or more of the body surface, are first treated for shock by morphine, external heat, vigorous administration of fluids and possibly by blood transfusion. As early as possible, gauze saturated with 2½% tannic acid solution is applied to the whole burnt area and kept moist with this solution for twenty-four hours or more until the burned skin is thoroughly tanned. The burned area is then treated by the open air method under a heated cradle, supplemented by warm boric acid or soda irrigations. The administration of fluids is pushed and epinephrin and sodium chloride may be given. Repeated blood transfusions are occasionally indicated. When all sloughs have disappeared and the wound is cleanly granulating, adhesive or rubber tissue strips are applied and "scarlet red" preparations may be used to accelerate epithelization. Skin grafting occasionally is advisable. For small burns, involving 5% or less of the body surface, the best treatment consists in the application of soothing ointments containing phenol or picric acid and gauze dressings.

### Plastic Rejuvenation Operations.

V. Lakatos (*Wiener Medizinische Wochenschrift*, May 5, 1928) reviews the work of Steinach and Voronoff regarding rejuvenation. Not being satisfied with the end results of these operations, he has devised one of his own. He makes ten to twelve incisions in the *tunica vaginalis*, thus enabling the testis to prolapse through the openings. According to the author the testis increases considerably in bulk and its functions are stimulated. This effect is noted immediately after operation, unlike similar effects following the methods of Steinach and Voronoff. Such operations on dogs have made them more active and eager to fight. Two males, aged sixty-seven and sixty-five, have been operated upon with definite increase in bodily vigour, muscular tonus, weight and appetite. They have been observed now for six months and the improvement shows no signs of diminution.

# British Medical Association News.

## SCIENTIFIC.

A meeting of the South Australian Branch of the British Medical Association was held at the Darling Building, University of Adelaide, on August 30, 1928, Dr. J. Corbin, the President, in the chair.

### Orthopædic Conditions.

Dr. L. O. Betts read a paper entitled: "Some Crippling Conditions and Their Treatment" (see page 488).

Dr. Malcolm Scott explained the mechanism of production of fracture of the carpal scaphoid. He said that it was due to the styloid process of the radius impinging on the neck of the scaphoid and chiselling it into two pieces. He had never seen this fracture unite by bone. The fracture he considered caused a varying disability. He showed the result of operative treatment in one patient.

As regards club foot he stated that it was supposed to occur in one in six thousand births. The parents were often told not to have treatment started until the child was three months old. As a matter of fact active treatment should be begun at birth and be finished by the time the child was three months old. He used adhesive plaster to retain the corrected position and changed this frequently at first. He used plaster of Paris only after an operation for tenotomy or a bone excision operation.

Of congenital dislocation of the hip, the diagnosis was easier when the lesion was unilateral. If both hips were dislocated, the proportion being one case in four, the appearances were more deceptive. The chief features were then the symmetrical waddle, the broad appearance of the pelvis and the raising of the great trochanter to the level of the anterior superior spine. He showed skiagrams illustrating the position in which the limbs were placed after reduction, in full abduction external rotation and flexion to a right angle. His custom was to leave the first plaster on for three months, then to reduce the position by 20% and put a second plaster for three months and to repeat the process again for three months, then allowing the child gradually to walk.

Dr. John Corbin agreed with the removing of a portion of a fractured scaphoid and he referred to a patient whose disability had been pronounced after successful replacement of the fractured fragment; the subsequent removal of a fragment had been necessary before complete relief of symptoms was obtained.

A large number of patients with backache *et cetera* who came for compensation, suffered from arthritis in the spine as shown by X rays and he considered that they should be guardedly informed of the presence of this, as they made it a means of not returning to work.

Deformities following rheumatoid arthritis with fixed flexed deformities were extremely crippling and in a large measure could be prevented by adequate treatment of the limbs during the acute stage by retention in the optimum position.

Sir Henry Newland said that, as regards Colles's fracture, complete reduction was essential, but opinions varied on the best type of splinting to be used. For the past ten years he had advocated placing the wrist in full flexion, pronation and ulnar deviation and maintaining this position by a piece of adhesive strapping over the heads of the metacarpals with its two ends adherent along the forearm. The extensor tendons acted as a splint for the fragments when they were thus put on the stretch.

He agreed with Dr. Scott in the mechanism of the production of fracture of the scaphoid and said that he had seen a case in which X ray examination showed that the tip of the styloid process of the radius had been separated as well as the fracture through the neck of the scaphoid. In dislocation of the semilunar bone the cause of the great pain which he considered undoubtedly a neuritis, was, he thought, due to compression of the median nerve which had been forced forward by the displaced bone and caught on the upper edge of the anterior annular ligament.

As to the causation of sciatica, he agreed with Putti's explanation in some instances. He said he had performed Jones's pseudarthrosis operation on three patients and considered that they might still have a stable limb without having to use a caliper. His three patients were able to walk comfortably without calipers.

Dr. H. Gilbert stated that he wished to emphasize three points. The first was the care which was necessary in the diagnosis of early tuberculosis of the hip in children. He quoted three patients who had come under his notice, all of whom had been treated for varying long periods with the vague diagnosis of "rheumatism." One child had been so treated for six months and had then been admitted to the Children's Hospital where an X ray examination had been made which revealed advanced tuberculosis of the hip. Eventually the child had died of meningitis. Another child had been treated for rheumatism for three months. A skiagram, taken then, revealed a little erosion of the head of the femur, but this had not been considered to be definitely tuberculous. This child had subsequently developed a tuberculous abscess. Another cause of mistaken diagnosis was an injury to the joint or an epiphyseal strain. Three weeks' rest from weight-bearing should cure this, if the diagnosis was correct. If the case went on longer it should be regarded with suspicion.

Dr. Gilbert's second point was that in regard to children suffering from congenital *talipes equino-varus* he had frequently been told by the parents that they had been advised that treatment was unnecessary until the child was a few months old or that the application of strapping was detrimental in a young child. He wished to stress the error in this and the absolute necessity for beginning the treatment for this condition at once after birth, as the position of the foot was mainly due to a maldevelopment of the astragalus. His method of treatment was to manipulate the foot into the correct position and to maintain it there by the application of a single piece of adhesive strapping.

Dr. Gilbert's third point was that in cases of paralysis following acute poliomyelitis it was essential to place the affected muscles immediately in a position of relaxation and to maintain them there by means of some form of splinting.

Dr. Bronte Smeaton said he had had recently a patient with an unusual fracture of the scaphoid in which the proximal fragment had been displaced backwards and had caused absorption of the margin of the radius where it impinged against it.

Dr. L. C. Lindon asked Dr. Betts, first, what further treatment he would advise in cases of stub injury to the shoulder joint when X rays revealed early arthritis which had been treated by rest in abduction without much evident improvement. Secondly, he asked the treatment of Kienbock's disease, osteitis of the carpal semilunar and thridly, he wished to know what Dr. Betts considered the cause of severe pain in the outer metatarsals in cases of *pes cavus*.

Dr. A. B. Jones asked if Dr. Betts had any experience of and if he knew of any objections to the reduction of a Colles's fracture with a local anæsthetic. He also pointed out the necessity for examining for associated injuries in the head of the radius and shoulder region.

Dr. Betts in reply said that union did occur in the majority of cases of fractured scaphoid if treatment was commenced early. The reason why lumbar arthritis of the type described had not been observed by radiologists, was that they had not been looking for pathological changes in this part of the lumbar spine, their attention being focused on the joints between the vertebral bodies. Furthermore, to show up the joints between the intra-articular processes it was necessary to take the radiograms with a fine focus tube as far as possible from the patient, with an exposure of thirty or forty seconds and a low kilovoltage. He congratulated Sir Henry Newland on his results from Jones's pseudarthrosis operation on the hip. Although some patients did not need a caliper, the majority required one to insure stability and lessen the tendency to increase of the shortening.

### Myositis Ossificans.

Dr. P. S. Messent showed a patient, thirteen years of age, a schoolboy, who had sustained an injury to his left elbow three years previously. No deformity of the bones

could be detected. X ray examination revealed a fracture of the lower end of the humerus with a fissure into the elbow joint. A week later slight movement and massage had been commenced. A fortnight later the elbow could be extended to 90° and a thickening had appeared in the region of the *brachialis anticus*. X ray examination had revealed ossification occurring in front of the humerus. Massage and movements had been stopped and the arm kept at rest in a sling. Dr. Messent considered it a case of *myositis ossificans*.

DR. J. A. BONNIN asked if the fracture had united and he was answered in the affirmative.

DR. L. O. BETTS said that he considered it a case of ossification in a blood clot, not a true myositis. He pointed out that Jones, of Liverpool, maintained that an injured elbow should be kept in flexion for three weeks and he thought that they had fewer stiff elbows than he had seen in Adelaide. He was not afraid that extension of the elbow would be limited by a formation of callus in the olecranon fossa, as, if it did form there, it was so soft that it soon became moulded and did not limit extension.

### Multiple Urinary Calculi.

DR. JOHN CORBIN reported a case of multiple calculi in the ureters and kidneys and showed the skiagrams.

### Growth of Bone.

PROFESSOR WOOLLARD explained a series of prints of skiagrams illustrating the growth of bone and of the epiphyses of the long bones which had been prepared for the Anatomical Department of the University by Dr. I. B. Jose, Honorary Demonstrator of Anatomy.

## MEDICO-POLITICAL.

A CIRCULAR notice is being forwarded to all members of the Victorian Branch of the British Medical Association concerning the General Practitioner's Section of the Branch. This section was formerly known as the Lodge Medical Officers' Section. An appeal is being made to all general practitioners to participate in the discussion on all matters affecting the welfare of the medical profession. It is stated that the gradual encroachment by governments and philanthropic institutions demands the watchfulness and education of every practitioner. It is only by frank discussion of the problems of lodge practice, national insurance, hospitals, health centres, ante-natal clinics, bush nursing and so forth that the views of general practitioners can be made known to the profession. It is hoped that these discussions will be of value to the Council of the Victorian Branch. The next meeting of the section will be held on October 19, 1928. Dr. R. H. Fetherston will read a paper on some points of the *National Insurance Bill* in its relation to the general practitioner.

# Correspondence.

## FEES FOR LIFE INSURANCE EXAMINATION.

SIR: I am directed by the Council of the Queensland Branch of the British Medical Association to draw attention to the following paragraph in the leading article of THE MEDICAL JOURNAL OF AUSTRALIA of September 1, 1928:

> The Branches and the Federal Committee have come to the conclusion that if a relatively high fee were charged, the societies would adopt other measures. Small policies can be accepted without examination; proponents for large insurances can be sent to whole-time referees. In these circumstances it has been determined that one guinea is the recognized fee for life insurance medical examination and report and that a fee of ten shillings and sixpence may be charged if no examination of urine is required and if the report is a short one.

I have also to request you to publish the following resolution of the Queensland Branch passed at a special general meeting of the Branch held on December 15, 1916:

> That the fee for all cases of life assurance be not less than one guinea.

Also the following resolution of the Federal Committee:

> That the Federal Committee recommends each Branch to fix one guinea as the minimum fee to be accepted for any life insurance examination and report, irrespective of the amount of the proposed policy and of whether or not the excretions are examined.

Yours, etc.,

E. S. MEYERS,
Honorary Secretary,
Queensland Branch,
British Medical Association.

Brisbane.
September 18, 1928.

[The resolution quoted above was adopted by the Federal Committee in April, 1917. Since then the matter has been discussed by the Committee in 1918, 1921 and 1928. Some Branches of the British Medical Association in Australia have found it advisable to retain the smaller fee of ten shillings and sixpence for an examination without analysis of the urine and report on a short form. At its recent meeting the members of the Federal Committee recognized the argument in favour of the smaller fee in the circumstances referred to and as a result the delegates of the South Australian Branch withdrew their motion to consider the question of an increased fee for examination and report.—EDITOR.]

# University Intelligence.

## THE UNIVERSITY OF MELBOURNE.

THE Beattie Smith lectures on insanity for this year are to be delivered at the Medical Society's Hall, East Melbourne, by Dr. R. S. Ellery. Dr. Ellery who is a medical officer in the Victorian Lunacy Department and holds the position of Clinical Assistant to the Psychiatrist at the Melbourne Hospital, has for several years been working on the treatment of cerebral syphilis by means of induced malaria. The subject of the lecture is "The Problem of Neurosyphilis and Its Treatment." Two lectures will be held at 8.15 p.m. on October 29 and November 5, 1928, respectively. The lecturer intends to present his subject chiefly from the psychiatric standpoint, with lantern and cinematograph illustrations. All members of the medical profession are invited to attend.

# Corrigendum.

DR. S. S. ARGYLE has called our attention to a mistake made in a leading article published in THE MEDICAL JOURNAL OF AUSTRALIA of September 29, 1928. Dr. Argyle was Minister of Health and Chief Secretary in the Lawson-Allan, the Lawson, the Peacock and the Allan-Peacock Ministries.

## A HOLIDAY EXPEDITION.

IN the past few years we have called the attention of our readers to the nature study expeditions which have been organized annually by Mr. E. F. Pollock, F.R.G.S., of the Royal Zoological Society of New South Wales and of the Royal Australasian Ornithologists' Union. While the two previous excursions have been to places outside Australia, the fifth expedition will be to the Kuringai Chase, National Park, Broken Bay and the Hawkesbury River. A camp will be pitched for a period of four weeks. Those participating will have unusual opportunities for nature study, will be able to enjoy daily water excursions in beautiful districts and safe bathing in enclosures and will be offered good sport, including shark fishing. A large pleasure boat has been chartered, capable of seating

upwards of eighty persons. The charge for the four weeks is twenty-five pounds. This includes everything; there will be no extras. We are informed that several ladies and gentlemen from States other than New South Wales have already joined the party. The party will leave Newport or Hawkesbury on December 28, 1928, and will return on January 24, 1929. Full particulars in an illustrated pamphlet can be obtained by application to Mr. Pollock, 7, Carrington Avenue, Strathfield, New South Wales. The excursion should be an ideal one and medical practitioners who can afford the time, will be well advised to consider the advisability of joining Mr. Pollock's party.

## Books Received.

STORY OF "SOUTHERN CROSS" TRANS-PACIFIC FLIGHT, 1928, by C. E. Kingsford-Smith and C. T. P. Ulm; 1928. Sydney: Penlington and Somerville. Crown 8vo., pp. 227, with illustrations.

METHODS AND USES OF HYPNOSIS AND SELF-HYPNOSIS: A TREATISE ON THE POWERS OF THE SUBCONSCIOUS MIND, by Bernard Hollander, M.D., M.R.C.S., L.R.C.P.; 1928. London: George Allen and Unwin, Limited. Crown 8vo., pp. 191. Price: 6s. net.

## Diary for the Month.

Oct. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Oct. 24.—Victorian Branch, B.M.A.: Council.
Oct. 25.—New South Wales Branch, B.M.A.: Branch.
Oct. 25.—South Australian Branch, B.M.A.: Branch.
Oct. 26.—Queensland Branch, B.M.A.: Council.
Nov. 1.—South Australian Branch, B.M.A.: Council.
Nov. 2.—Queensland Branch, B.M.A.: Branch.
Nov. 6.—Tasmanian Branch, B.M.A.: Council.
Nov. 7.—Western Australian Branch, B.M.A.: Council.
Nov. 8.—Victorian Branch, B.M.A.: Council.
Nov. 9.—Queensland Branch, B.M.A.: Council.
Nov. 13.—Tasmanian Branch, B.M.A.: Branch.

## Medical Appointments.

Dr. R. L. Park (B.M.A.) has been appointed Deputy Quarantine Officer, Melbourne.

* * *

Dr. David Duncan Cade (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for the Insane at Mont Park, Victoria.

* * *

Dr. Albert Curtis (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for the Insane and the Receiving House, Ballarat, Victoria.

* * *

Dr. Alan Thomas Britten Jones has been appointed Acting Honorary Assistant Surgeon at the Adelaide Hospital.

* * *

Dr. Garton Maxwell Hone (B.M.A.) has been appointed Honorary Anæsthetist at the Adelaide Hospital.

* * *

Dr. Alexander Jamieson Meikle has been appointed Acting Superintendent, Parkside Mental Hospital, South Australia.

## Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

Einasleigh District Hospital, Queensland: Medical Officer.

Launceston Public Hospital: Resident Medical Officers (2).

Maryborough Hospitals Board, Maryborough, Queensland: Junior Resident Medical Officer.

Repatriation Commission: Visiting Specialist, Junior Resident Medical Officer.

## Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

| BRANCH. | APPOINTMENTS. |
|---|---|
| New South Wales: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney. | Australian Natives' Association.<br>Ashfield and District United Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham United Friendly Societies' Dispensary.<br>Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney.<br>Marrickville United Friendly Societies' Dispensary.<br>People's Prudential Benefit Society.<br>Phœnix Mutual Provident Society. |
| Victorian: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing.<br>Brisbane United Friendly Society Institute.<br>Stannary Hills Hospital. |
| South Australian: Secretary, 207, North Terrace, Adelaide. | All Contract Practice Appointments in South Australia.<br>Booleroo Centre Medical Club. |
| Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia. |
| New Zealand (Wellington Division): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales.

## Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to "The Editor," The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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